THE American JOURNAL OF Surgery

MAY 1955

THURSTON SCOTT WELTON, M.D., EDITOR

Surgical Review
Tumors of the Neck
Bronchopulmonary Diseases

SIXTY-FOURTH YEAR OF PUBLICATION

MAY 1955

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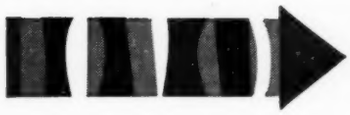

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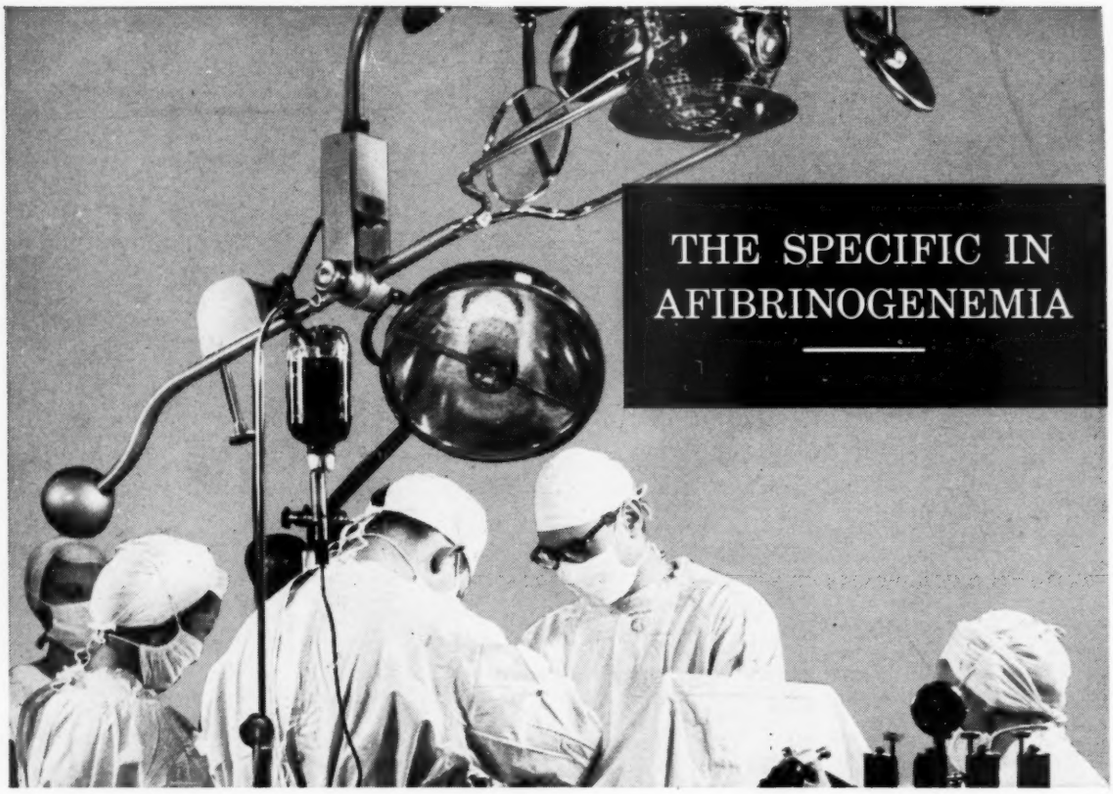

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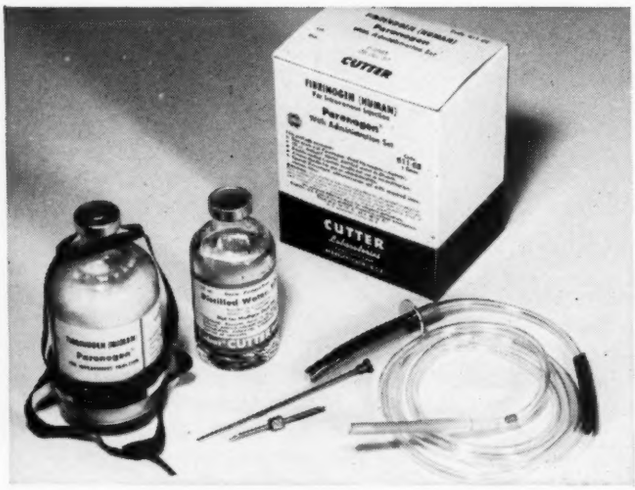

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IRVING ROSENBERG AND PHILIP V. SKERRETT

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A. DAVID SCHWARTZ

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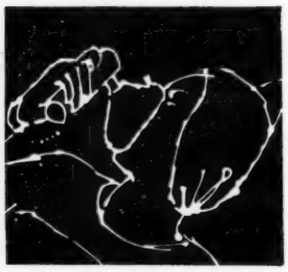

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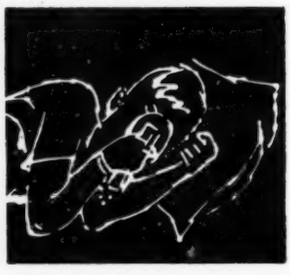

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FRANCIS A. BENEVENTI AND FRANCIS S. CREIGHTON

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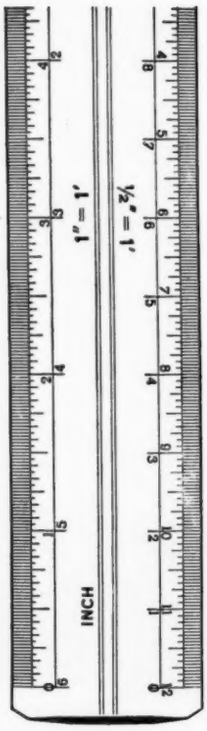

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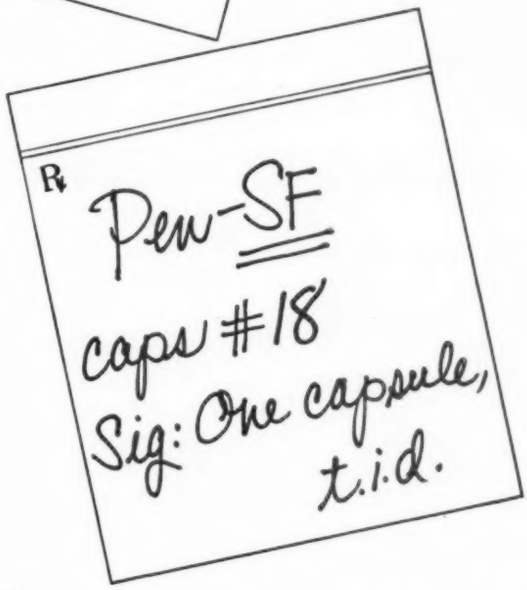

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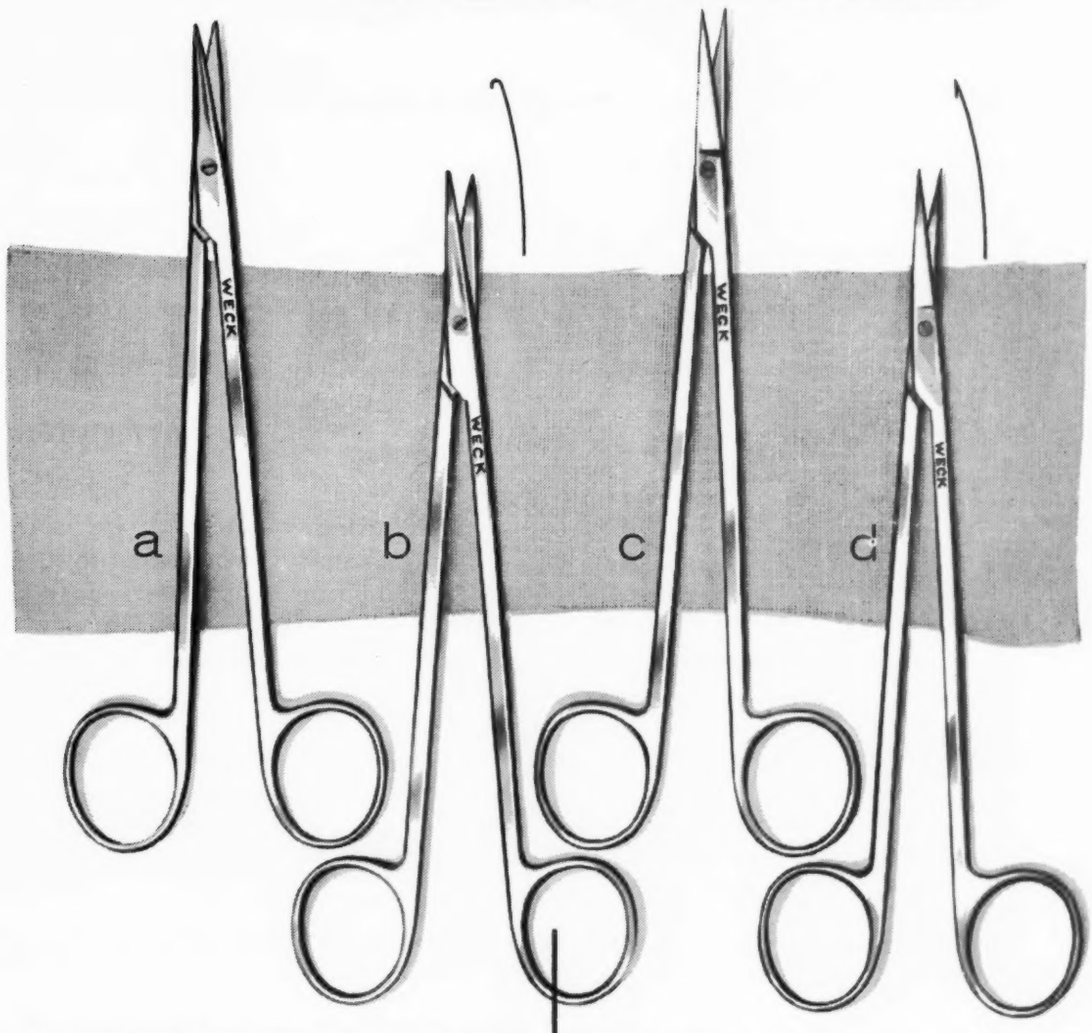

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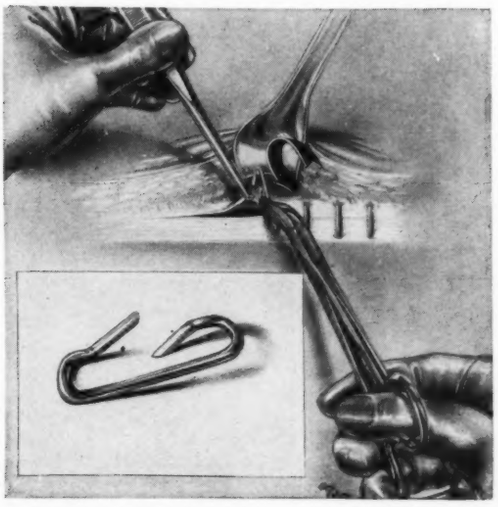

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By Paul I. Hoxworth, M.D.

Cincinnati General Hospital

Clinical and experimental observations having shown that flexibility of suture material is, in some respects, undesirable, and that a rigid wire suture is superior to flexible materials for preventing wound disruptions, a new method of abdominal wound closure has been developed.

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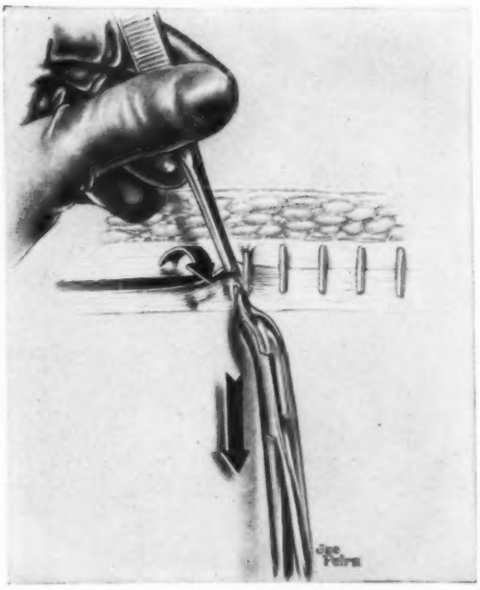

The Hoxworth technic, the Fashure and the special applying forceps are described in detail and fully illustrated in the new issue of *The Armamentarium,* Volume II, Number IV. Be sure you read of this important new method of avoiding wound disruption. If your copy of The Armamentarium has not yet arrived, a note or card of request will bring it to you promptly.

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1. Beck, W.C. and Collette, T.S.: Editorial—False Faith in the Surgeon's Gown and Surgical Drape: Am. J. Surg., 83:125-126, 1952.
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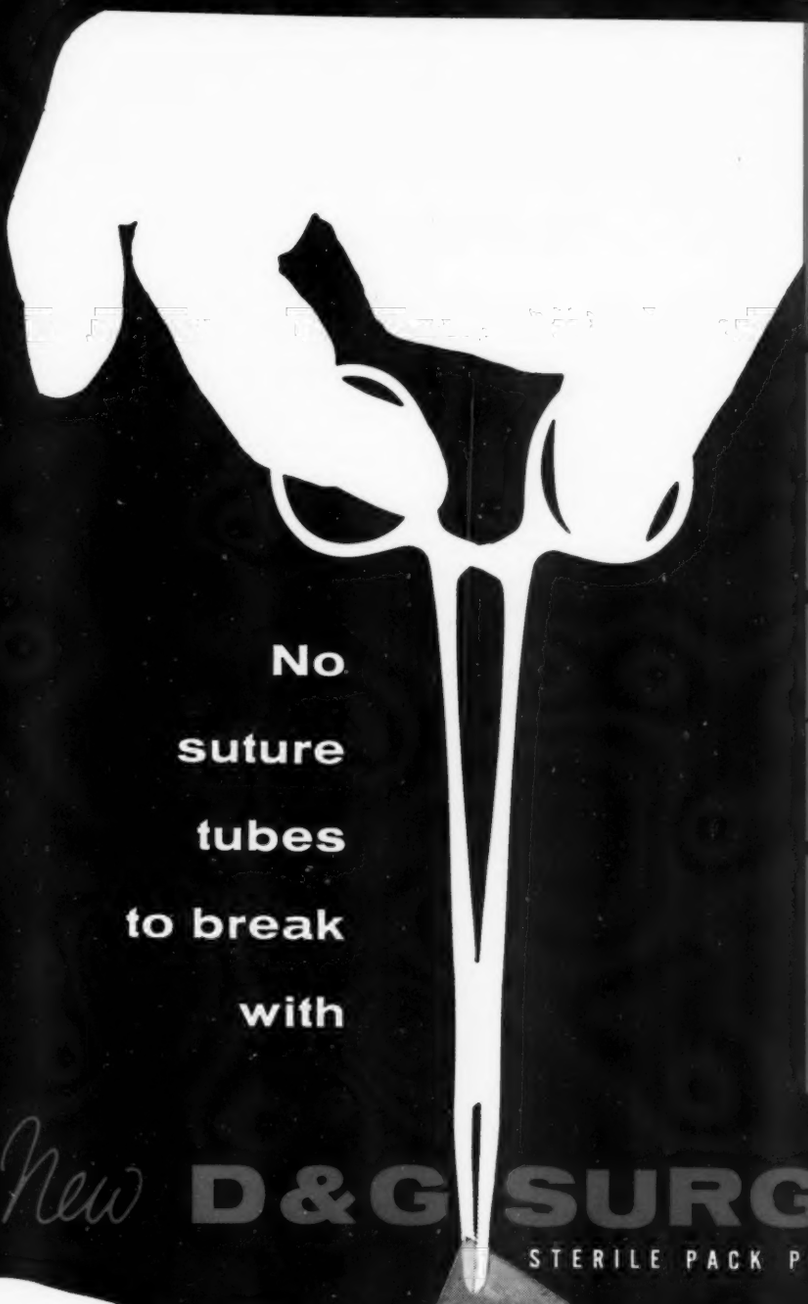

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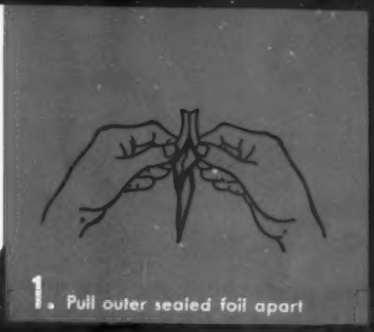

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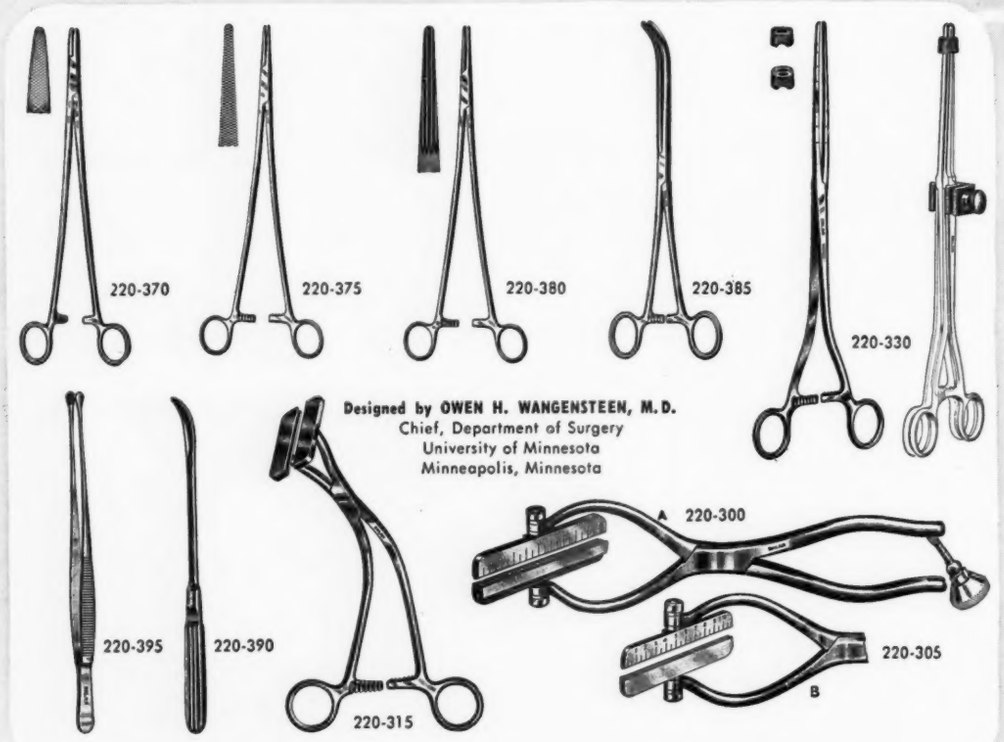

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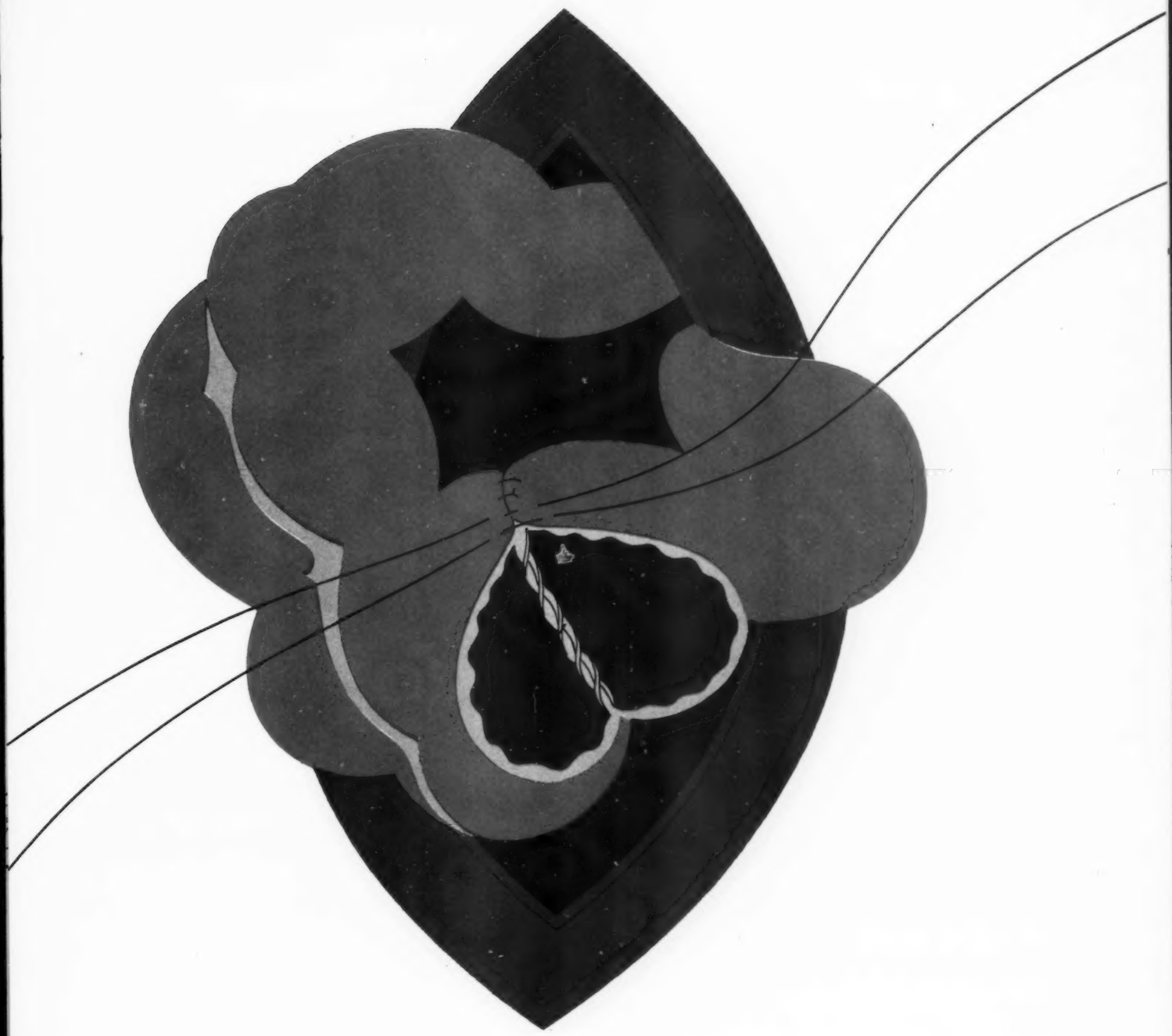

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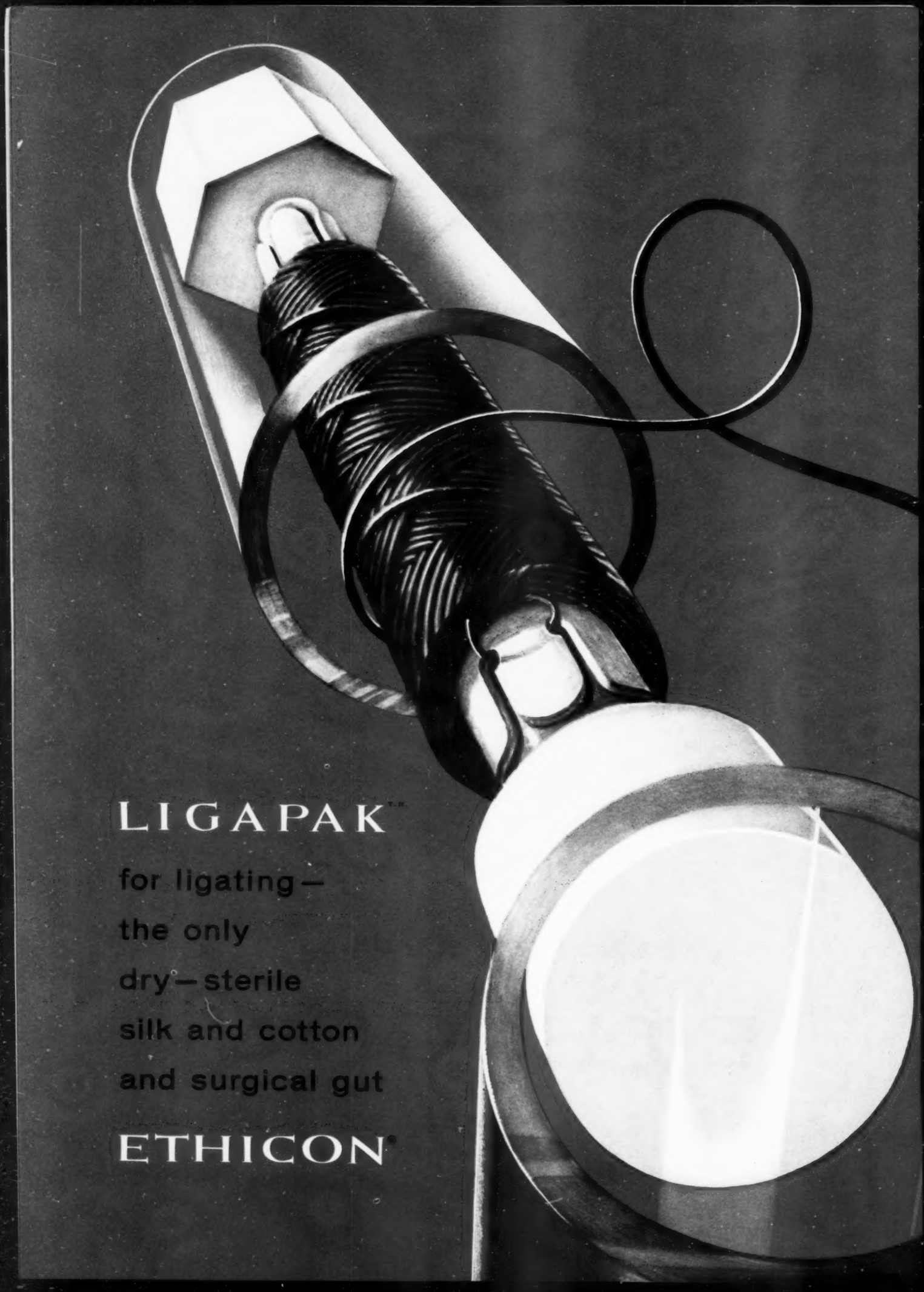
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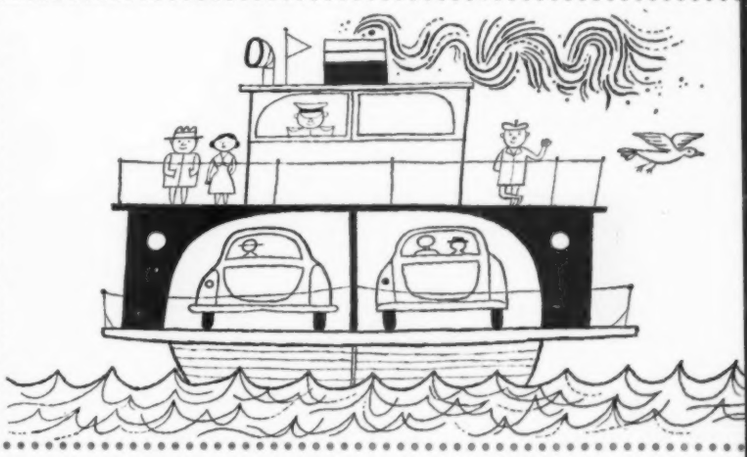

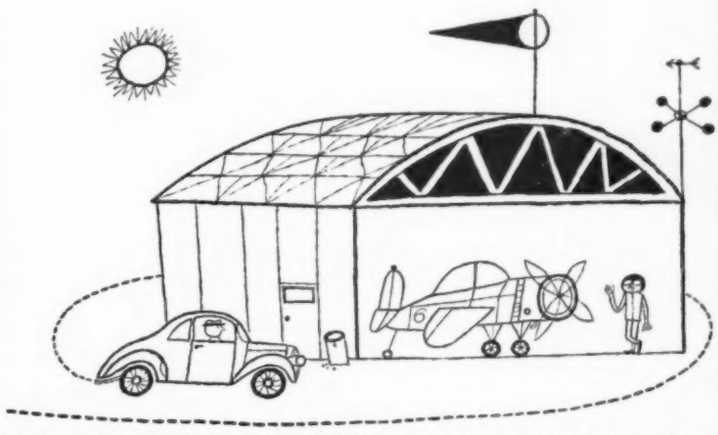

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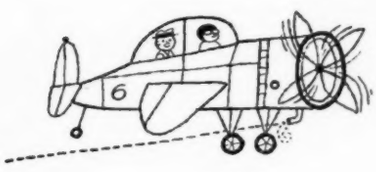

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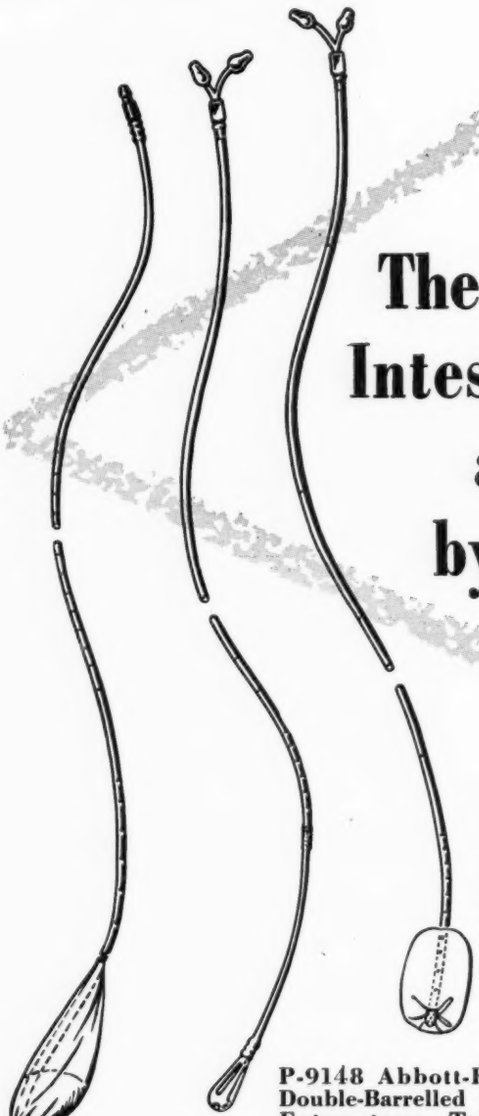

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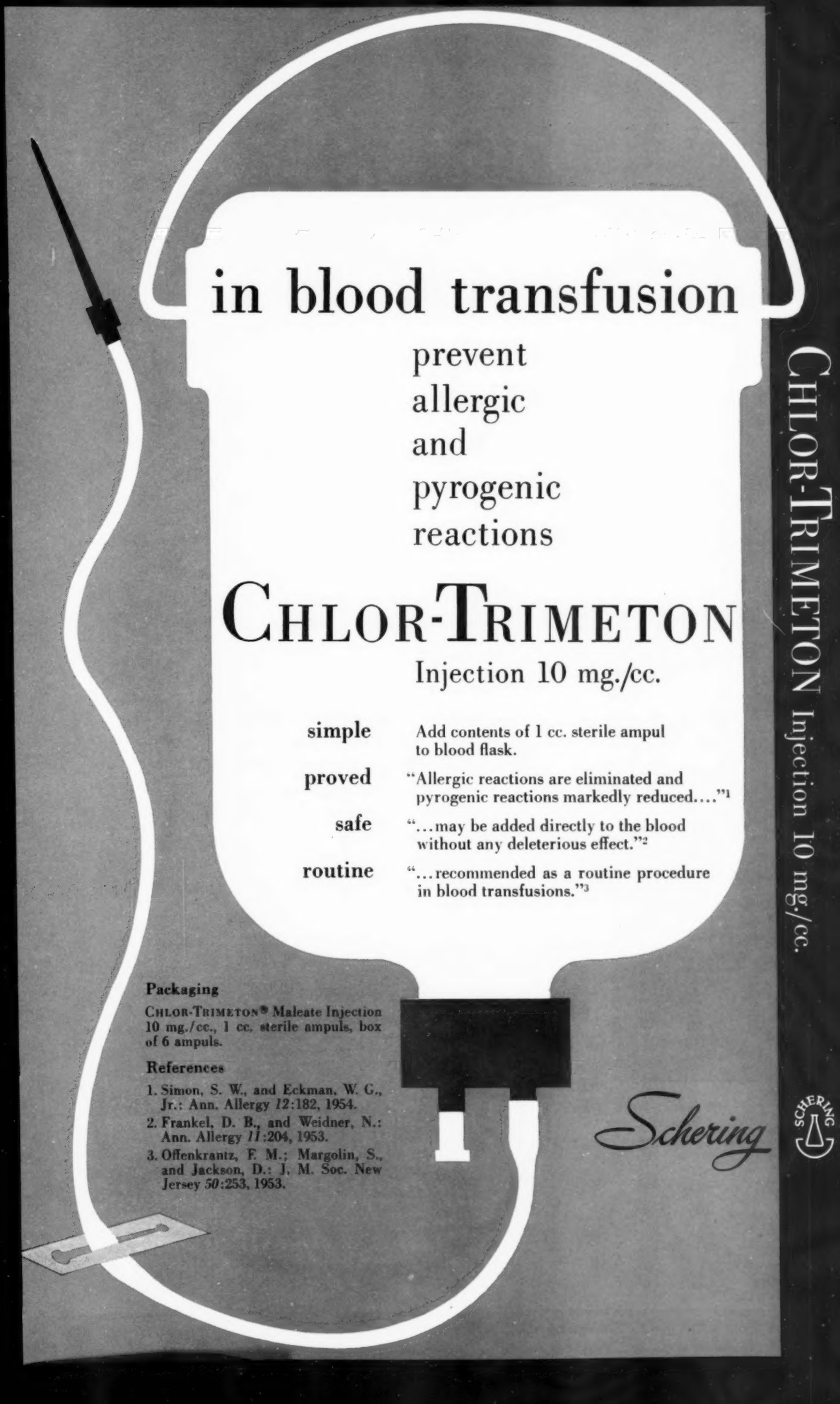

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1. Simon, S. W., and Eckman, W. G., Jr.: Ann. Allergy *12*:182, 1954.
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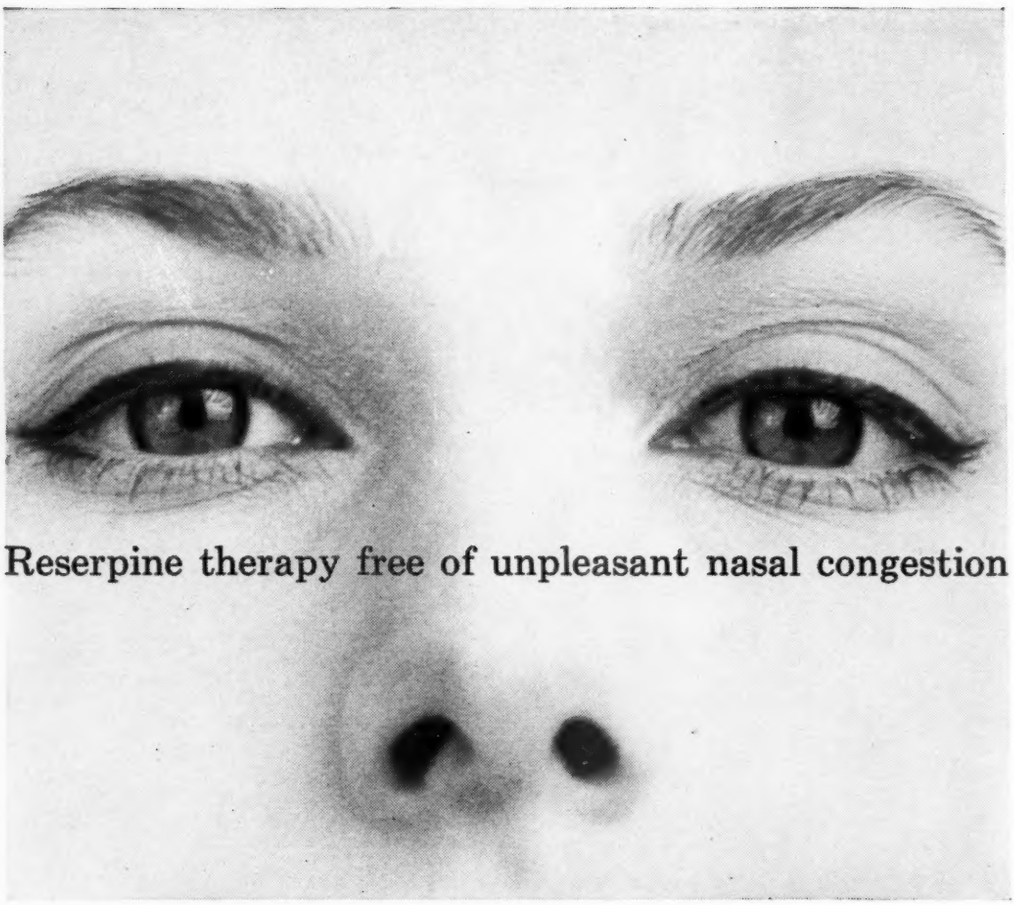

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Editorial

A CRITICAL ANALYSIS OF THE TREATMENT OF CARCINOMA OF THE BREAST

WHEN one considers that more than 50,000 women die each year of cancer of the breast in this country, it behooves the medical profession to reconsider and evaluate this problem often. Emphasis on the need for more careful appraisal of our past and present accomplishments has been appearing with increasing incidence in the current literature. There has been a decided change in attitude about the validity of the traditional radical mastectomy, and I believe that we should cast aside the fervent desire to achieve better statistical results and accomplish more surgical triumphs. Instead, we should seriously entertain ideas and methods that will accomplish more good for a greater number of patients.

Perusal of the recent literature and conversations with many surgeons reflect the increasing discouragement with the results of radical mastectomy. The various attempts to extend the surgical procedures in order to eradicate disease is obviously proof of the fact that the traditional radical mastectomy must have failed in the treatment of this disease. The mere fact that these extended procedures can now be done almost with impunity because of our improved methods of anesthesia and our better understanding of physiologic processes does not justify their indiscriminate use. A careful analysis of the underlying circumstances that have made the radical mastectomy a failure will prove that the ultra-radical procedures should not be continued without the most serious evaluation of the facts.

The premise upon which the radical mastectomy was originally performed was the belief that cancer of the breast would spread, as a rule, in one direction, i.e., into the axillary nodes. The work of numerous investigators has proved this to be completely erroneous, and that the pathways for both venous and lymphatic dissemination are many and variable. The lesion can spread to the supraclavicular region without involving the axilla; it may spread to the internal mammary nodes and mediastinum, directly into the chest, over to the other axilla and breast, into the abdomen and even down to the inguinal nodes. The spread of the disease can therefore be considered unpredictable. Moreover, the microscopic spreading edge of the disease cannot be determined clinically by the operating surgeon, so that the most aggressive type of surgery is never assurance of the eradication of all disease. However, it will produce morbidity and mortality which might be considered prohibitive.

Three rather recent articles have illustrated many points worthy of repetition, indicating the futility of the radical treatment of many of the breast lesions predestined at the time to be lethal. Of particular interest is the fact that the numerous modes of tumor spread have to a large extent been overlooked because of an overemphasis on lymphatic spread. This has been based on the frequent misconception that the lymphatic system is an autonomous circulatory system until it reaches communication

with the major blood vessels. We often forget that the blood and lymphatic systems at the point of tissue fluid movements are closely related and this probably explains the all too frequent case with a small lesion, no regional adenopathy and with distant metastases.

The location of the lesion in the breast and the prediction of the involvement of a particular group of nodes is another point for emphasis. Handly and Thackery have indicated that the internal mammary chain of nodes is involved in a high percentage of malignant lesions of the breast. Other investigators have proposed that the internal mammary group of nodes is more likely to be involved with a lesion in the medial quadrant of the breast, and have devised ultra-radical operations to eradicate all suspected involved tissue. On the other hand, Hutchinson has found no correlation between the location of the lesion in the breast and the incidence of mammary node involvement in the study of eighty-one consecutive cases.

The scope and extent of operation has been evaluated by many. A most interesting recent report by Mayer and Smith indicated that in a group of 148 cases of cancer of the breast treated in a small hospital the patients who were operated upon by the more inexperienced group of surgeons who did procedures that fell far short of the recognized radical mastectomy, did better and have a more satisfactory five-year survival rate than those patients operated upon by the more expert surgeons performing radical mastectomy. This of course is no reflection on those who perform radical mastectomy or an indictment of their surgical skill. However, it does seem to indicate that perhaps the more extensive procedures may be conducive to more rapid dissemination because more avenues for spread of disease are opened by those procedures. Mayer and Smith report a five-year survival rate of 57 per cent for patients who had simple mastectomy, while the rate for the patients with radical mastectomy was 49 per cent.

Many open-minded surgeons have realized the futility of all the heroic attempts and now have been willing to consider the simpler methods of therapy. Although surgery is without doubt the best method available for certain very early localized lesions, the simpler procedures with adequate postoperative radiation therapy seem to be a modality for treatment which has been proved to be adequate and statistically, at least, as good as the radical mastectomy, without the postoperative morbidity and mortality that follow the radical procedures. Reports in support of this are appearing in the literature with increasing frequency.

This change in trend of thinking about the surgery for cancer of the breast is not new, for Halsted himself must have realized the inadequacy of the radical mastectomy. It was not long after the publication of this method of treatment that he himself attempted supraclavicular and mediastinal extensions of the operation. However he, very wisely, soon afterward did not continue the use of the ultra-radical measures. I daresay that had Halsted been alive today he would probably even discontinue the use of the operation that bears his name.

I do not understand the violent negative attitude of some brilliant men in this field toward use of the simpler procedures. Surely it must be some reason other than the lack of knowledge about the progress of this disease regardless of what type of therapy is used.

Cancer of the breast has been considered incurable by many observers; and certainly if we cannot cure the patient, let us employ a modality of therapy that will do the least harm. I sincerely believe that simple mastectomy and possibly the removal of accessible axillary nodes, with a high standard of adequate postoperative radiation therapy, should be considered the procedure of choice in the treatment of this disease.

Adequate proof is at hand that certain forms of cancer can be transiently restrained in human beings by the use of chemotherapy. We may thus hope for a future, perhaps not too far distant, when chemicals will be available which will be as restraining of one or the other form of cancer as certain antibacterial agents are today for one or other bacterial strain. Certainly our surgical efforts have been exhausted and have proved to be a failure in a very large percentage of the total group of women that apply for treatment. There is, indeed, obvious reason for the dilemma of the conscientious surgeon.

THOMAS C. CASE, M.D.,
New York, New York

Diagnosis of Tumors of the Neck

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HARRY C. SALTZSTEIN, M.D., *Detroit, Michigan*

From the Surgical Service of the Harper Hospital, Detroit, Michigan.

TUMORS of the neck have their own diagnostic problems. One does not have to be concerned with complicated and atypical indigestion histories, interpretation of x-ray films, functional tests, physiologic imbalance, and the other multitudinous details of abdominal diagnosis. In neck lesions the history, except for exophthalmic goiter, is brief. The objective information obtained by physical examination takes but a few minutes. There are no outstanding laboratory aids (again excluding goiter). One must know the diagnostic possibilities and the roster of various disease entities appearing in the neck.

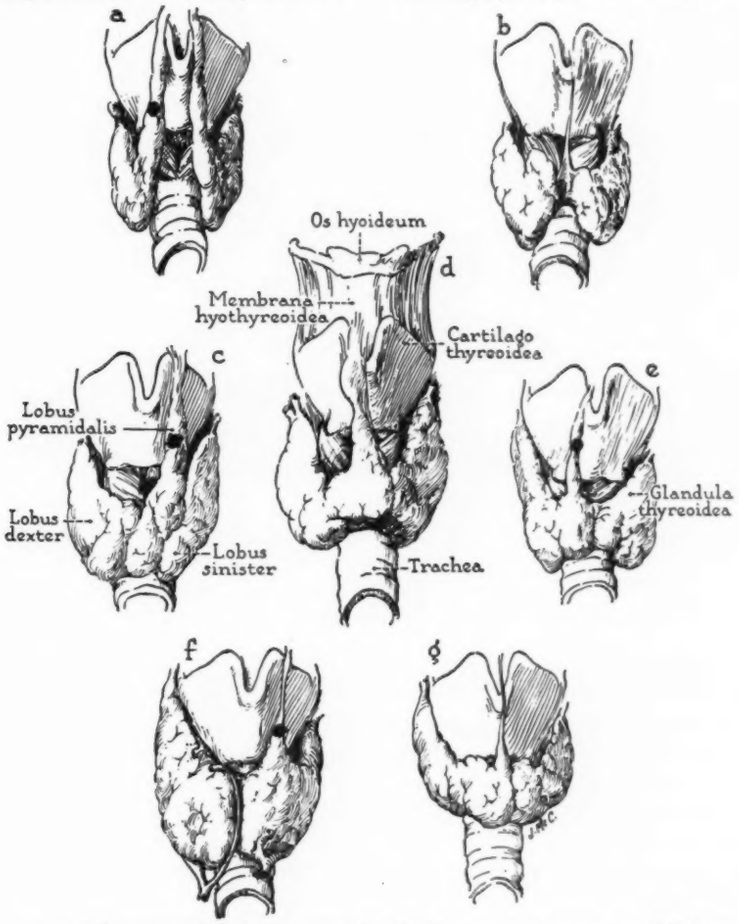
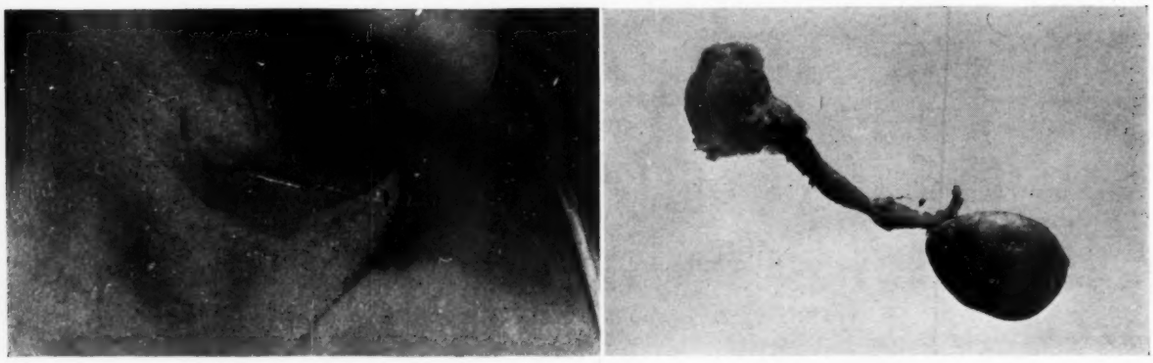


FIG. 1. The pyramidal lobe; note that in four out of seven possible locations, the pyramidal lobe is not in the midline. (From ANSON, B. J. Atlas of Human Anatomy, Philadelphia, 1950. W. B. Saunders Co.)

The diagnosis of neck conditions during the past decade or so has had two important de-



2A 2B

FIG. 2. A, thyroglossal cyst in a thirty-eight year old woman; tumor is 4 to 5 cm. from the midline; was present one year. B, specimen; rounded nodule with stalk proceeding obliquely upward and medially to mid-portion of hyoid bone; specimen includes excised mid-portion of hyoid bone.

3 4

FIG. 3. Thyroglossal cyst in a man aged fifty-eight. Soft fluctuant mass in region of left lobe of thyroid; several weeks' duration. This had been diagnosed as thyroid adenoma. On excision it proved to be an infected thyroglossal cyst.

FIG. 4. Sebaceous cyst. Exactly in the position which the usual thyroglossal cyst presents; no tract to hyoid bone palpable subcutaneously.

velopments: (1) Now we rarely have exophthalmic goiter to consider as a diagnostic possibility and (2) we are seeing more older patients with, as a corollary, an increased incidence of those diseases which affect older patients, namely, metastatic carcinoma.

There are various classifications of neck diseases. One used by Lahey was: (1) conditions found in the midline, (2) single discrete masses in the lateral portions of the neck and (3) multiple masses in the lateral neck regions. However, there are not very many midline conditions, and many lesions which start in the lateral portion of the neck may be either single or multiple.

This discussion will briefly list and illustrate: thyroglossal cysts, papillary adenocarcinoma of the thyroid (Graves' disease and the usual adenomas and carcinomas of the thyroid will be excluded), miscellaneous benign conditions, parotid tumors, carotid body tumors, lymphomas and metastatic carcinomas. Emphasis will be on the anatomic site.

THYROGLOSSAL CYSTS

Although all texts (so far as the author knows) state that thyroglossal cysts appear in the midline, a chart in Ansons' Atlas of Human Anatomy published in 1950 (Fig. 1), shows seven illustrations of the pyramidal lobe of the thyroid, and in four of them the pyramidal lobe is depicted lateral to the midline. This means

that thyroglossal cysts do not always appear in the midline. Figure 2A illustrates a tumor which appeared in a thirty-eight year old woman as far lateral as the border of the sternomastoid muscle and below the hyoid bone. When dissected, it was a solid thyroid adenoma, but on excision proved to be a typical thyroglossal cyst. (Fig. 3.)

Figure 4 shows a sebaceous cyst in the classical anatomic location for a typical thyroglossal cyst.

Dermoids and dermoid cysts may also appear in the midline. They sometimes depress the floor of the mouth or obstruct the trachea, either above or below the larynx or in the superior mediastinum.

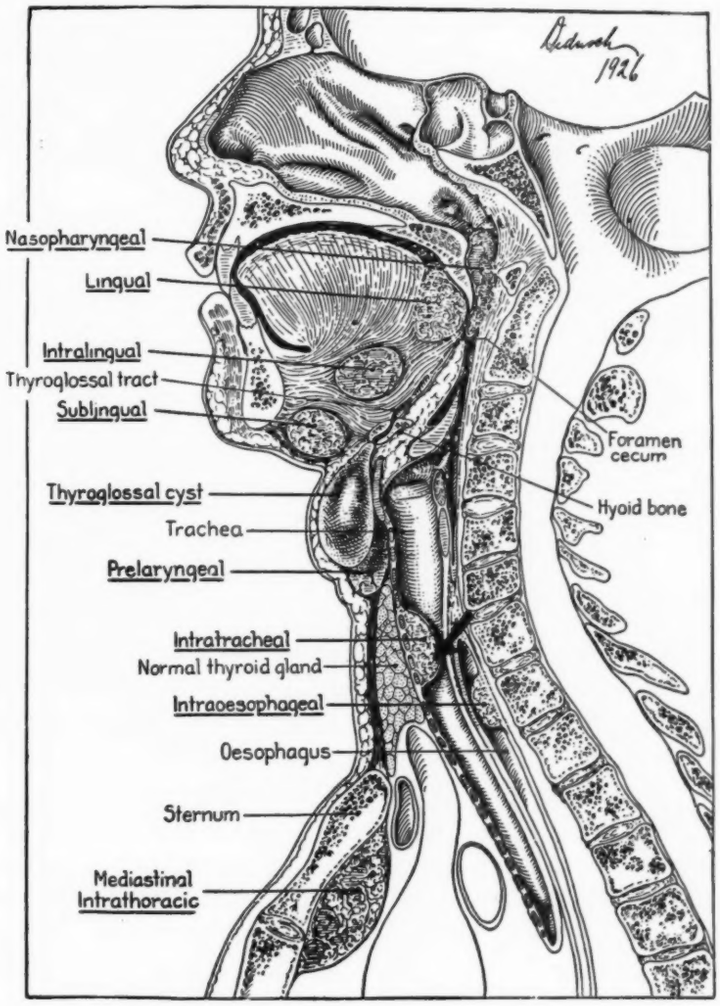


FIG. 5. Regions in which aberrant thyroid tissue may occur in the midline; arrow points to location of lesion in Figure 6. (From Lewis' Practice of Surgery, vol. 6, chap. 1, page 229. Edited by WALTMAN, W. Hagerstown, Maryland. W. F. Prior Company.)

nodule and the specimen (Fig. 2B) shows a 4 cm. tract going obliquely to the midline and ending just beneath the mid-portion of the hyoid bone. Another case (a fifty-eight year old man) presented a soft semi-fluctuant mass in the region of the left thyroid lobe distinctly lateral to the midline. There was a history of an acute onset with slight fever, then gradual development of the mass over a period of several weeks. It had been diagnosed thyroid

PAPILLARY ADENOCARCINOMA OF THE THYROID

Everyone is familiar with lingual goiter and the danger of hypothyroidism if the goiter is removed. It may be the only thyroid tissue the patient has. An old edition of Dean Lewis'

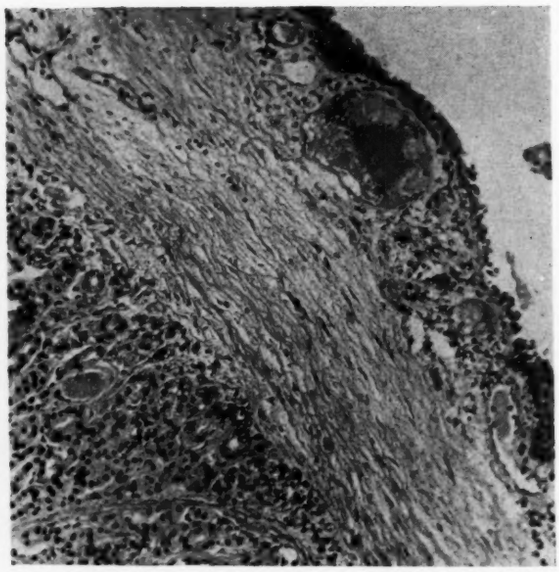


FIG. 6. Papillary adenocarcinoma of thyroid; nodule of trachea underneath epithelium in a nineteen year old girl. Note normal trachial mucous membrane on right and papillary adenocarcinoma of the thyroid on left which shows irregular papillary formations and colloid. Patient was well five years after local removal through tracheotomy. (Courtesy of Dr. Wyman D. Barrett and Dr. Lyle Waggoner.)

Practice of Surgery shows thyroid tissue aberrant in almost any location along the midline from the nasopharynx to the mediastinum, including the tongue, pharynx, trachea and esophagus. (Fig. 5.) The diagnostic significance of these various aberrant thyroid masses is that most of them have been shown within the last decade or so to be carcinomas metastatic from a primary source in the thyroid gland.

Figure 6 shows a nodule of carcinoma of the thyroid located underneath the epithelium of the trachea in a fifteen year old child. Following thyroidectomy, the patient almost suffocated in the recovery room. An emergency tracheotomy disclosed a large subglottic mass. It was excised locally and the patient has been well for five years.*

Another illustration from the same volume of Lewis' Practice of Surgery (Fig. 7) depicts the aberrant thyroid tissue in the submaxillary triangle, at the edge of the trapezius muscle, or even far out in the supraclavicular region. If one superimposes markers indicating that these masses may appear underneath the borders of the sternomastoid muscle, more especially along the jugular chain going down toward the mediastinum, one will have a picture of the possible lateral locations of papillary adenocarcinoma of the thyroid. The adenocarcinoma,

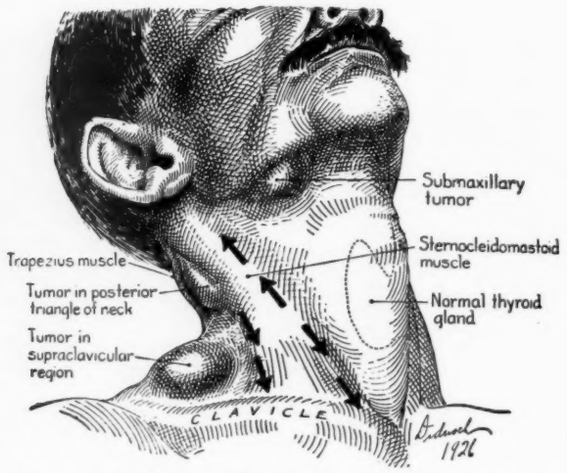


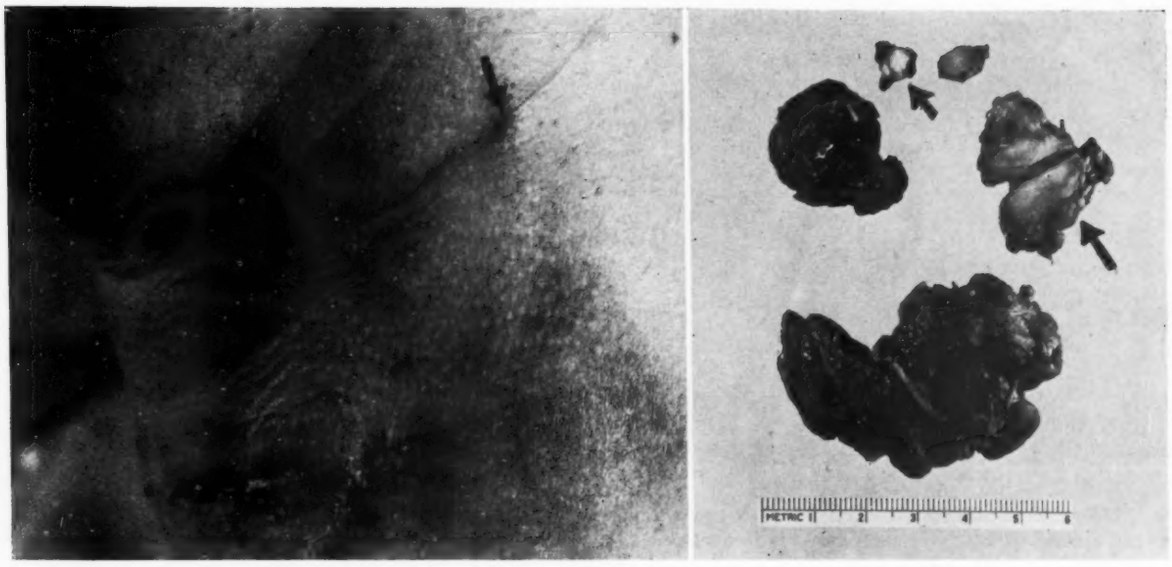
FIG. 7. Possible lateral locations of aberrant thyroid tissue. (Modified from Lewis' Practice of Surgery, vol. 6, chap. 1, p. 230. Edited by WALTMAN, W. Hagerstown, Maryland. W. F. Prior Company.)

then, may appear anywhere in the lateral regions of the neck.

All of these tumors are metastatic, originating from a primary source in the thyroid gland. Very often the original focus is minute and microscopic. The files of several institutions, which had had large numbers of these cases, invariably revealed that further sections of the thyroid showed small carcinomas. These nodules appear often in young women, sometimes in adolescents. Usually one can feel nothing but the nodule; the thyroid response to palpation is negative. They are more common than one might think; and if the diagnostic possibility is kept in mind, they are not so confusing. A single lozenge-shaped or ovoid mass (sometimes multiple masses), lying laterally along the border of the sternomastoid or beneath it, may not be a branchial cyst or Hodgkin's disease but may be a low-grade carcinoma originating in the thyroid gland.

A small nodule underneath the posterior border of the sternomastoid at the hyoid level had been observed in a patient for eight or nine months by an internist. Finally an x-ray therapist, to whom she had been sent, advised its removal. It proved to be carcinoma of the thyroid. (Figs. 8A and B.)

* Case to be reported in detail by Dr. Lyle Waggoner, Detroit, Michigan, courtesy of Drs. W. D. Barrett and Lyle Waggoner.



8A 8B

FIG. 8. A, papillary adenocarcinoma of thyroid in a forty year old woman; arrow shows where a 2 cm. tumor was removed; lower scar is the subsequent thyroidectomy incision; nothing abnormal was palpable here. B, thyroidectomy specimen; arrows show papillary adenocarcinoma which was in lower left pole of thyroid gland; smaller nodules were located exactly where left recurrent laryngeal nerve entered larynx. Patient was well ten years after local removal of neck nodule and thyroidectomy.

FIG. 9A. Papillary adenocarcinoma of thyroid in a fourteen year old girl; actual distribution of tumors; no symptoms; local removal of involved masses.

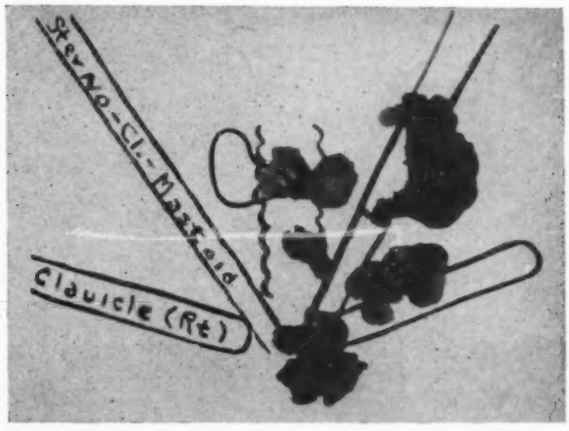
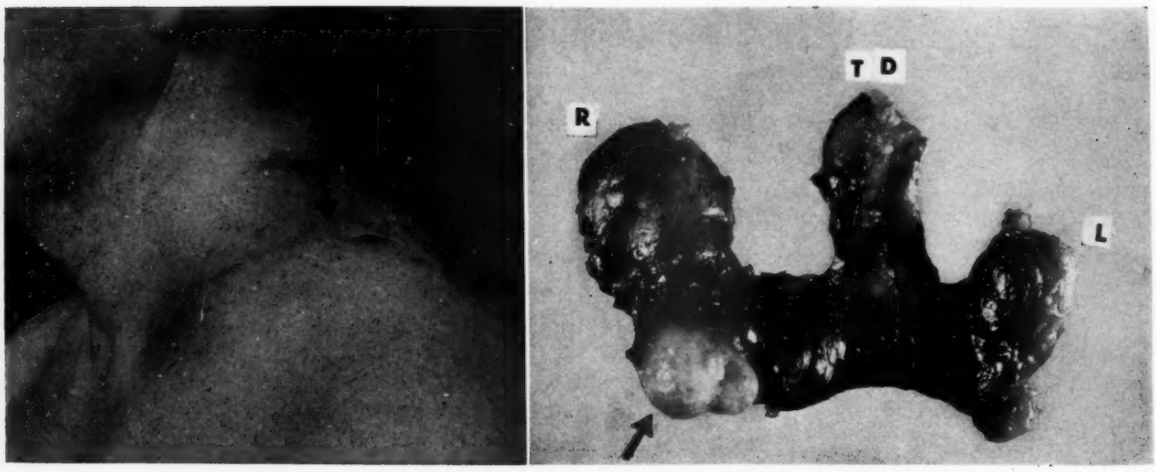


FIG. 9B. Gross specimens removed; no block dissection. Patient is well after eight years.

Figures 9A and B show multiple masses in the lateral portion of the neck in a fourteen year old girl. There were no symptoms; the child was in perfect health except for very large masses (indicated by the markings on the photograph). The distribution is typical—along the esophagotracheal groove, along the jugular chain and carotid sheath, in the supraclavicular region and down toward the superior mediastinum.

These tumors may also be bilateral and even may appear below the clavicle in the axilla. As indicated in Figure 7, they may appear far out in the supraclavicular region. Figure 10A is a photograph taken postoperatively of a fifty-six year old man who presented a mass arising just behind the lateral end of the clavicle near the acromion and in front of the scapula. On exploration this mass was located in front of the anterior surface of the scapula and behind the brachial plexus. Figure 10B



10A 10B

FIG. 10. A, papillary adenocarcinoma of thyroid in fifty-four year old man; postoperative photograph; mass presented far out in left lateral supraclavicular region in front of scapula. See Figure 7. B, thyroidectomy specimen. Note papillary adenocarcinoma in right (opposite) lower pole of thyroid.

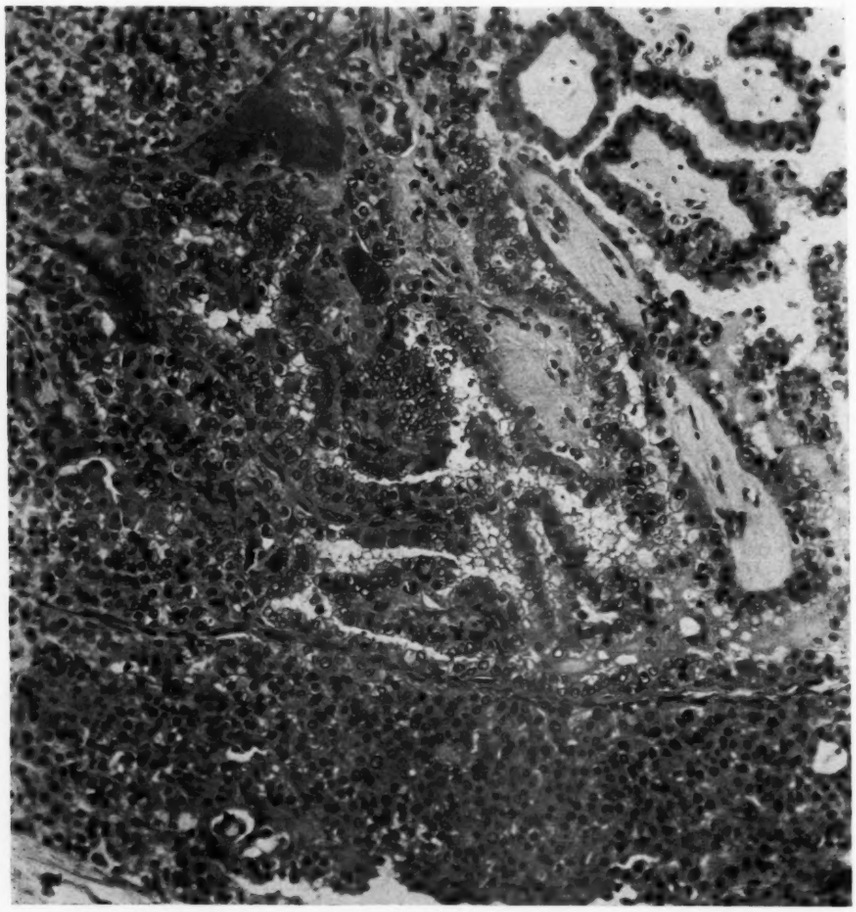
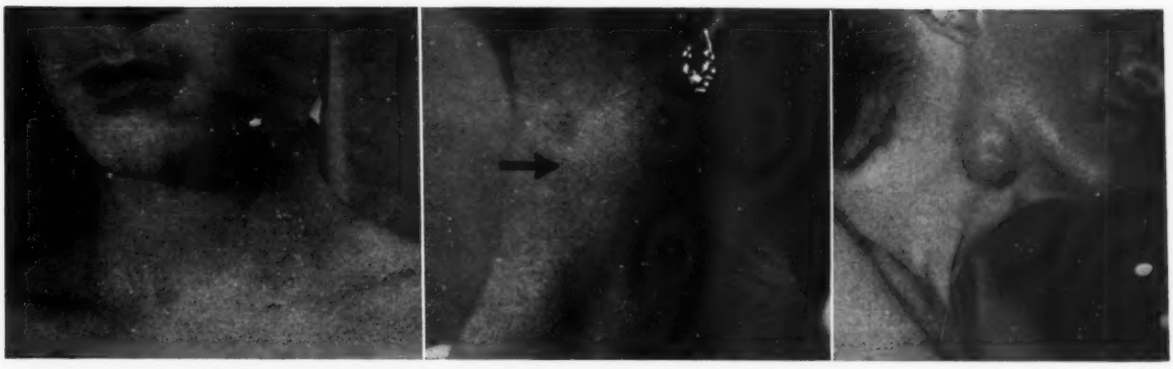


FIG. 11. Papillary adenocarcinoma of thyroid; usual microscopic appearance; low cuboidal cells in irregular columns with colloid in irregular acini; low grade malignancy.



12 13 14

FIG. 12. Simple adenitis in thirteen year old boy; thought to be possible lymphoma; subsided spontaneously in three weeks.

FIG. 13. Hyperplastic lymphadenitis.

FIG. 14. Chronic tuberculous abscess in submaxillary region; patient (forty-seven year old man) had had previous draining sinuses in posterior neck region; had atypical pulmonary infection which ultimately proved to be tuberculosis.

shows the primary tumor in the lower pole of the right (opposite side) of the thyroid gland.

Pathologically (Fig. 11), these tumors are all quite similar. They are well encapsulated, slowly growing, low-grade papillary adenocarcinomas with very little heaping up of the layers of endothelium, with colloid visible in some of the acini. Generally, the tendency of the carcinoma to metastasize is slight. In two of twenty-one patients, metastasis occurred within five years.*

Several authorities (though not all) believe that local excision of all of the masses is sufficient and that, because they rarely invade muscles or blood vessels, it is not necessary to do a radical dissection. Of the ten or twelve patients which I have followed, all have remained well following local removal of the masses. No radical neck dissections for papillary adenocarcinoma of the thyroid have been performed by the author.

LATERAL BENIGN LESIONS

Table 1 is a catalogue of the different benign conditions that may be found in the lateral portions of the neck. Inflammation can start anywhere. A simple adenitis from a sore throat (Fig. 12) may be confused with a lymphoma or Hodgkin's disease until it subsides after two or three weeks. Hyperplastic adenitis may persist for several weeks and may again be confused with either tuberculosis or branchial cysts before subsiding spontaneously. There was no way of telling whether the mass shown in Figure 13 was tuberculosis, a branchial cyst or papillary carcinoma of the thyroid. It proved, on excision, to be a hyperplastic lymph node. Figure 14 shows a chronic tuberculous abscess, so rare nowadays that it is scarcely thought of in the differential diagnosis.

TABLE 1
MISCELLANEOUS BENIGN LESIONS OF THE NECK (LATERAL)

Inflammation:	*Cysts, Benign Neoplasms, etc.:*
Acute	Branchial cyst
Hyperplastic adenitis	Lipoma
Chronic	Sebaceous cyst
Granuloma, etc.:	Dermoid cyst of neck
Tuberculosis	Chondroma
Lues	Osteoma (mandible)
Boeck's sarcoid	Hemangiomas
Lupus erythematosus	Lymphangiomas
German measles	Cirsoid aneurysm
Mumps	Neurofibromas
Actinomycosis	Schwannoma
Chronic brucellosis	Neurosarcoma
Infectious mononucleosis	Hygroma cysticum colli
Calculus:	*Diverticulum:*
Submaxillary gland calculus	Esophageal diverticulum

The patient had atypical pulmonary tuberculosis, plus several abscesses on the nape of the neck; tubercle bacilli were not positively diagnosed until three years after onset of the disease.

Inflammation of the salivary gland, secondary to a calculus in Wharton's duct (Figs. 15A and B), can mimic a tumor of the sub-

* CRILE, G., JR. Diseases of the Thyroid, p. 249. Philadelphia, 1949. W. B. Saunders.

15A 15B

FIG. 15. A, salivary calculus with chronic inflammation of submaxillary gland in thirty-five year old man. B, salivary calculus with chronic inflammation of submaxillary gland in man, age thirty-five.

16A 16B

FIG. 16. A, branchial cyst. B, branchial cleft sinus in five year old boy. Small sinus present since birth low in neck just medial to anterior border of sternomastoid; intermittent drainage of droplet of mucoid material; opening would admit only finest filiform probe; thick firm cord of the tract palpable for 2 cm. upward; removed through stepladder incisions. Sinus was easily followed: went above crotch of carotid bifurcation, underneath digastric muscle, to lateral pharyngeal wall.

maxillary gland or carcinoma metastatic from the mouth. The anatomic locations are identical. (Fig. 32A.) The gland may swell only after meals or subside after several weeks. X-ray will show the calculus caught in the mouth of the submaxillary salivary cut.

Figure 16A shows a typical branchial cyst. The chief confusion is with tuberculosis which, as indicated, is not frequent today. Several years ago I watched one of these patients for two years under the diagnosis of tuberculosis. I repeatedly aspirated some thin turbid fluid, and the patient received x-ray therapy. When the x-ray therapist went on vacation, his substitute suggested that the mass be removed It proved to be a simple branchial cyst which was dissected easily. Unless there is marked chronic inflammation, the branchial cyst can be easily removed. Figure 16B shows a branchial cleft sinus present since birth in a five-year old child. The sinus goes in a typical fashion between the crotch of the bifurcation of the carotid, underneath the digastric and into the pharynx anterior to the tonsillar region. The entire tract must be traced completely or there will be recurrence.

FIG. 17. Lipoma in front of right ear, region of anterior portion of parotid, in fifty-five year old man. Lipomas can occur anywhere in the face or neck region, and may be underneath the deep fascia.

FIG. 18. Dermoid cyst; loculated dermoid cyst in submaxillary region. (From *S. Clin. North America*, 33: 650, 1953.)

Superficial tumors such as lipomas can easily be confused with parotid tumors when they appear on the side of the face in front of the ear. (Fig. 17.) Lipomas may lie underneath the deep cervical fascia and ramify underneath the muscles or vessels; because the enveloping fascia is tense, they may feel like a deeply placed solid tumor. They must be dissected out completely or they will recur.

A sebaceous cyst, such as that shown in Figure 4, may be confused with parotid tumors, branchial cysts or any other superficial laterally placed mass.

Dermoid cysts usually appear in the midline. Figure 18 from the Lahey Clinic illustrates a loculated dermoid cyst in the submaxillary region.

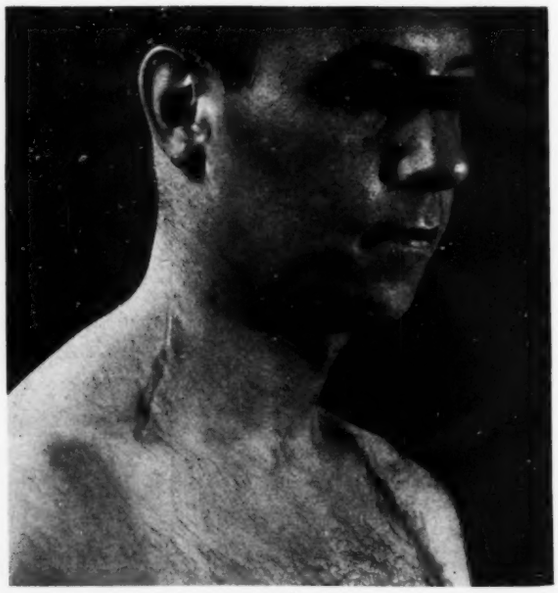
Any of the tissues in the neck may give rise to a tumor. Hemangiomas and lymphangiomas (Fig. 19A and B) come from blood vessels. Neurofibromas, schwannomas, and neurosarcomas may come from nerve elements. (Fig. 19 C and D.)

Usually the diagnosis is evident if one bears these multiple conditions in mind. However, as indicated in the introduction, there are cases in which it is impossible to make the differential diagnosis. Then the diagnosis is simply a lump which should be removed.

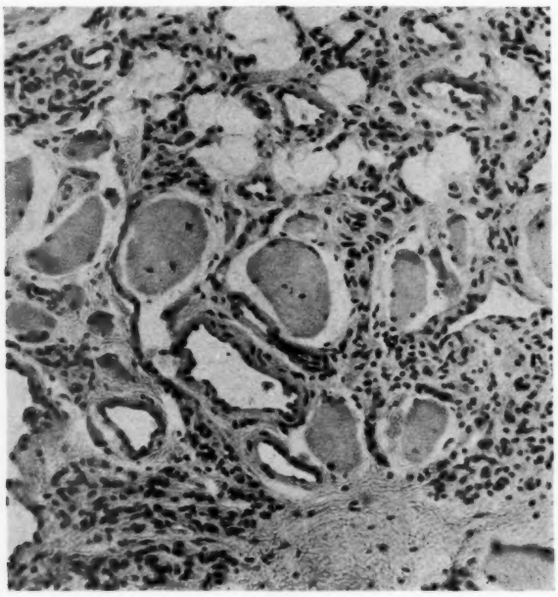
It should be kept in mind that squamous cell carcinoma (Fig. 20) or basal cell carcinoma (Fig. 21) can originate anywhere in the neck or face, and that the head and neck are frequent sites of origin for melanoma. (Fig. 22.) Melanoma in this situation brings its own problems. If well developed, a very wide radical removal, including a complete radical neck dissection, is indicated. Even then the prognosis is dubious.

TUMORS OF THE PAROTID GLAND

Tumors of the parotid gland show the central mass at the point of the lobe of the ear, pushing the ear lobe outward. (Fig. 23.) However, they may appear anywhere over the surface of the parotid gland, either posterior to the ear at the angle of the jaw, or along the anterior surface of the gland at the edge of the hairline. In this latter situation especially, they may be confused with superficial lipomas (Fig. 17) or sebaceous cysts. Many of them are still removed in doctors' offices under local anesthesia on the assumption that they are simple benign and very superficial tumors. Reports from such institutions as Memorial Hospital show that two-thirds of their cases of parotid gland tumors are recurrent carcinomas of the parotid gland which have been previously removed elsewhere. All recent



19A



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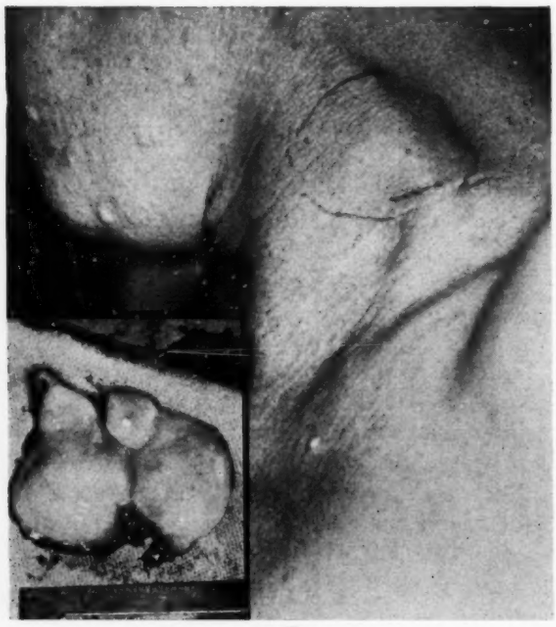

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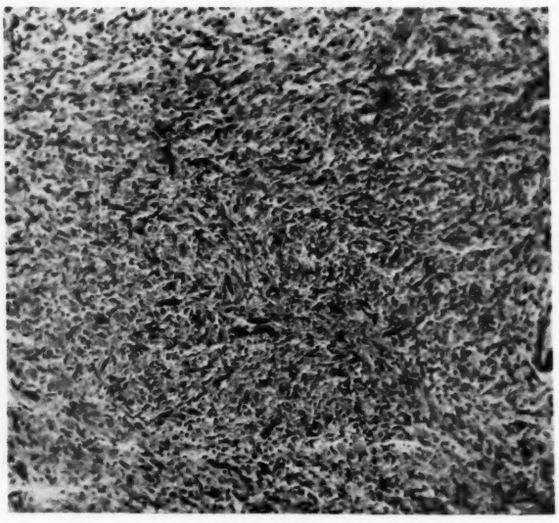


19D

FIG. 19. A, lymphangioma; small subcutaneous tumor in supraclavicular region in front of trapezius muscle. This is a very poorly placed incision; the spinal accessory nerve courses just in front of trapezius muscle in this location; if injured, wing scapula may ensue. Patient was a twenty-eight year old man. B, microscopic appearance; large spaces filled with lymph and lined by single layer of flat endothelium. C, recurrent neurofibrosarcoma in a thirty-nine year old woman; exploration six months previously (note scar) with diagnosis of fibrosarcoma. Patient presented 4 by 4 cm. tumor behind sternomastoid muscle at level of larynx. Resection of 5 by 5 cm. soft, greyish, homogeneous, well encapsulated tumor which extended into the space between sixth and seventh transverse processes and was intimately associated with upper cords of brachial plexus. Residual partial paralysis of upper cords of brachial plexus. Note lesions on lower face; had similar von Recklinghausen skin lesions over trunk. D, microscopic appearance, neurofibrosarcoma.

FIG. 20. Squamous cell carcinoma of skin of neck.

FIG. 21. Recurrent basal cell carcinoma of chin.

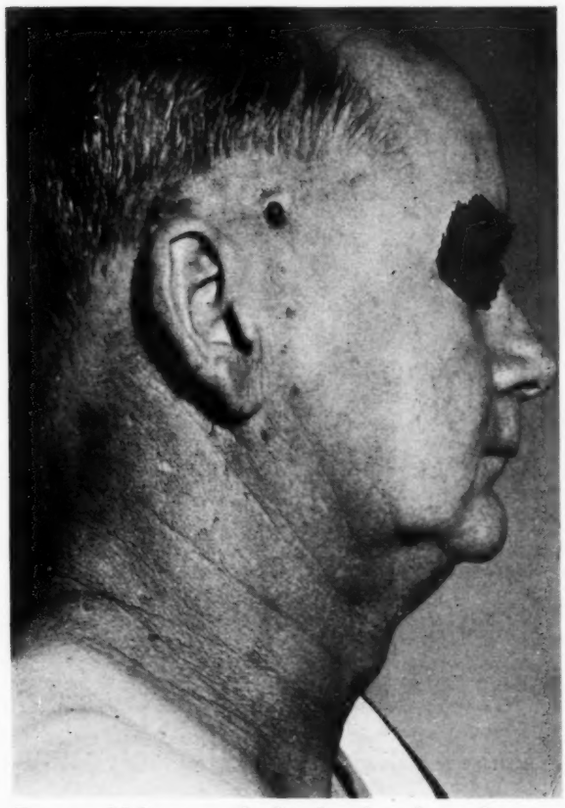


FIG. 22. Melanoma; the head and neck are frequent sites of origin for melanomas. (Courtesy of Dr. Clifford D. Benson.)

FIG. 23. Mixed tumor of parotid gland; note tip of lobe of ear in center of tumor; male, age forty.

reports agree that unless the tumor is superficial the facial nerve should be exposed after it leaves the skull, deep to and just above the mastoid eminence, before the tumor is resected. Only then can a wide enough margin of the parotid gland plus the capsule of the tumor be removed to prevent recurrence.

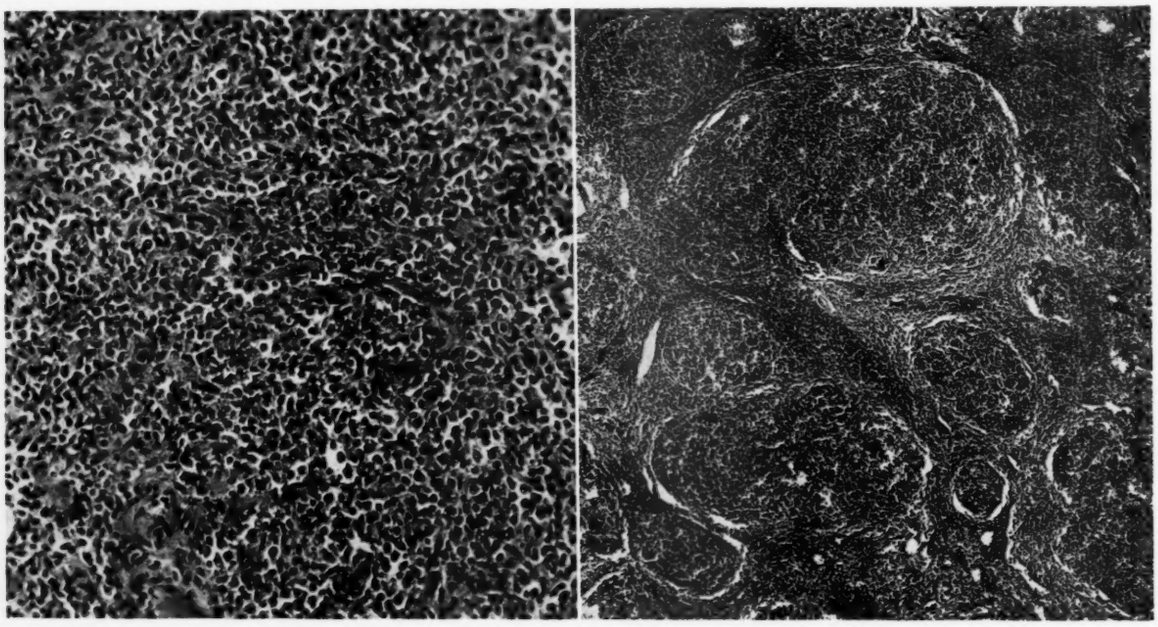
Ten to 15 per cent of salivary mixed tumors occur in the submaxillary salivary gland. If one does not see many neck tumors, a mixed tumor of the submaxillary gland is a rarity. (Figs. 24A and B.)

LYMPHOMAS

Perhaps the most diagnostically confusing type of tumor which we find in the neck is the lymphoma. It is confusing to the surgeon and to the pathologist. The author has records of cases in which several of the best pathologists in the United States have differed in the diagnosis of a lymphoma, some calling a particular microscopic section chronic inflam-

24A 24B

FIG. 24. A, mixed tumor of the submaxillary salivary gland in a forty-three year old man. B, specimen.



25 26

FIG. 25. Reticulum cell sarcoma; diffuse field of large single delicately stained cells arising from the primitive reticulum cell in the center of the node.

FIG. 26. Giant follicular hyperplasia (Brill-Symmers disease); large follicles, sometimes distinguishable with the naked eye and frequently covering two or three low power fields.

mation, others Hodgkin's disease and still others lymphosarcoma.

The four usually described entities are: (1) reticulum cell sarcoma (stem cell sarcoma or clasmatocytic lymphoma); (2) giant cell follicular hyperplasia or lymphoma (Brill-Symmers disease); (3) lymphosarcoma (small cell or lymphocytic and large cell or lymphoblastic lymphoma), and (4) Hodgkin's disease (lymphogranuloma). The microscopic picture is clear cut and easily diagnosed. Reticulum cell sarcoma (Fig. 25) shows a diffuse field of simple large cells with single, large, delicately stained nuclei and a large amount of pink-staining cytoplasm. It is a sarcoma arising from the primitive central reticulum of the lymph gland. It is rare and perhaps the most rapidly fatal of the lymphomas, death frequently occurring within six months. The giant follicular hyperplasia (Fig. 26) shows a large

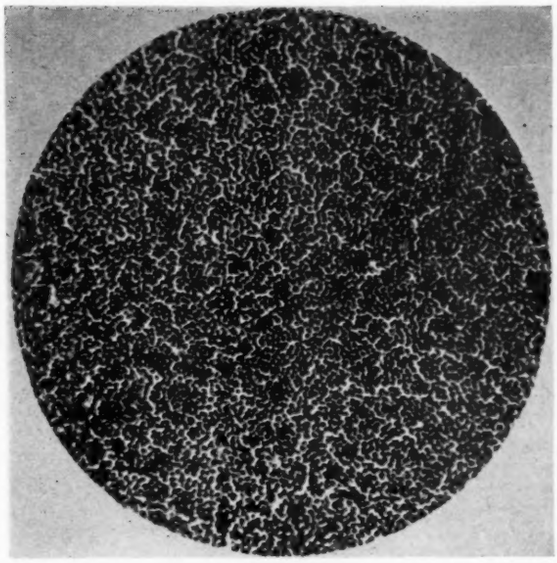


FIG. 27. Lymphosarcoma; solid sheet of dark staining mature lymphocytes. × 200.

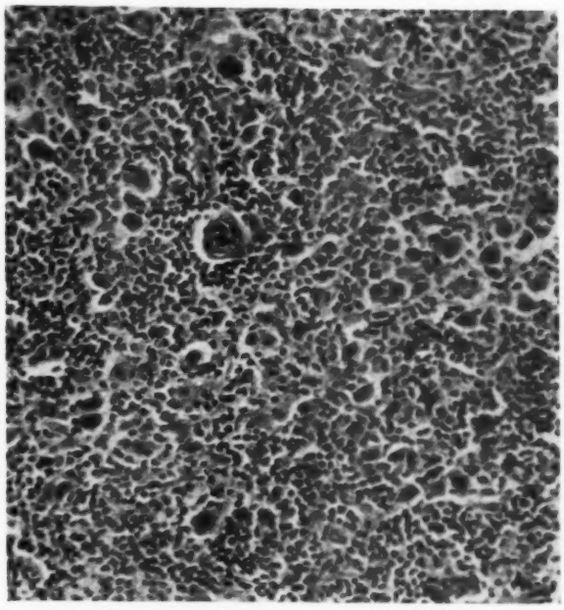


FIG. 28. Hodgkin's disease; variety of different types of cells; lymphocytes, plasma cells, fibroblasts, eosinophils; Dorothy Reed giant cells. × 225.

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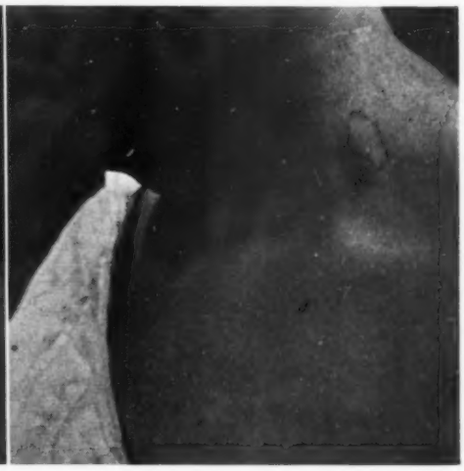


30

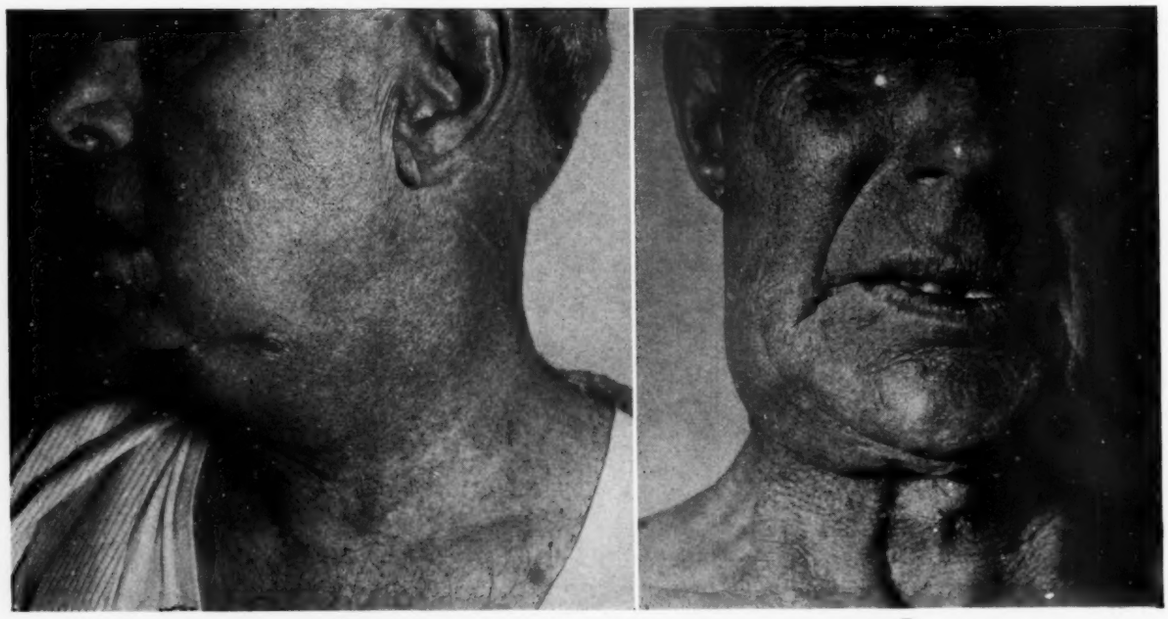
FIG. 29. Hodgkin's disease in twenty-six year old woman. Similar nodule on opposite side.

FIG. 30. Lymphosarcoma; single nodule; man, age forty-four.

follicle often covering two or three low power fields; often it can be seen with the naked eye. Perhaps half of these will cause no further trouble after a small amount of x-ray treatment, but others develop into lymphosarcoma or Hodgkin's disease which have serious prognosis.*

The problem is one never knows which patients will stay well and which lesions will progress. The typical lymphosarcoma (Fig. 27) is a solid sheet of mature black-staining lymphocytes, all of the same size with no stroma whatever and no follicle structure remaining. Hodgkin's disease (Fig. 28) is often confused with atypical inflammatory conditions. The inflammations will subside and Hodgkin's disease probably will not. The microscopic picture is often very confusing. There is a variety of different types of cells: monocytes, lymphocytes, neutrophils, eosinophils, plasma cells, fibroblasts, various degrees of pink-staining stroma and Sternberg-Reed giant cells. Lymph node

* One recent report shows 20 per cent five-year arrested cases.



31A 31B

FIG. 31. A, oral cancer. Metastatic spread from cancer of the lip in a sixty-four year old man; primary spread in the neck to the nodes posterior to the submaxillary gland. Exception: when the primary is directly in the midline of the lower lip; the submental nodes are then the first involved. B, carcinoma of the right lower lip near right angle of mouth had been treated by x-ray therapy one year previously; was gone in two or three weeks, no local recurrence; man, age sixty-six. Nine months later mass was noted right side of face; extensive involvement of right mandible and right submaxillary region in stony fixed metastatic malignancy; so-called "watermelon mass."

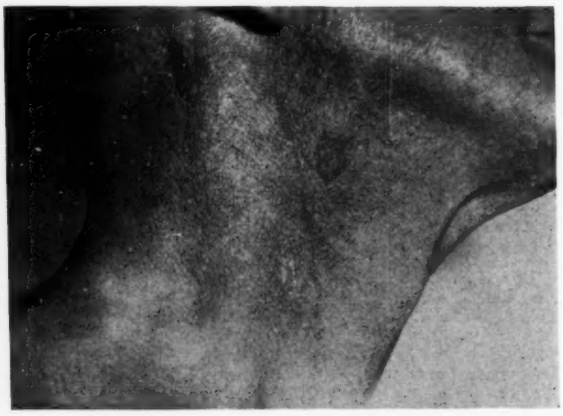


FIG. 32A. Oral cancer; metastatic spread from primary in lateral border of tongue; man, age fifty-eight; nodes below the digastric are usually the first involved; node was definite but palpation had to be done carefully to find it.

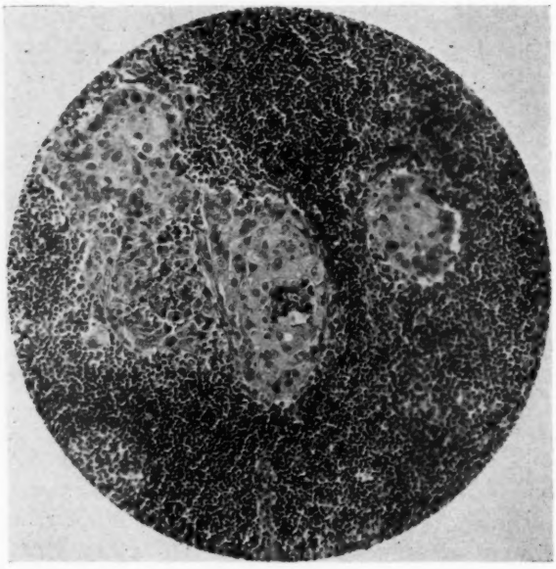


FIG. 32B. Microscopic appearance, squamous cell carcinoma invading the central portion of the node.

structure is destroyed and the capsule is involved. Some textbook statements indicate that one should not diagnose Hodgkin's disease without the giant cell; however, most pathologists will do so if they see the profusion of the various cell elements previously mentioned. In spite of the difference between Hodgkin's disease and lymphosarcoma, many cases are indistinguishable clinically. Figure 29 shows a small nodule in the right side of the lateral neck triangle in a twenty-six year old woman. There was a similar nodule on the right side. It proved to be Hodgkin's disease. Figure 30 was a similar

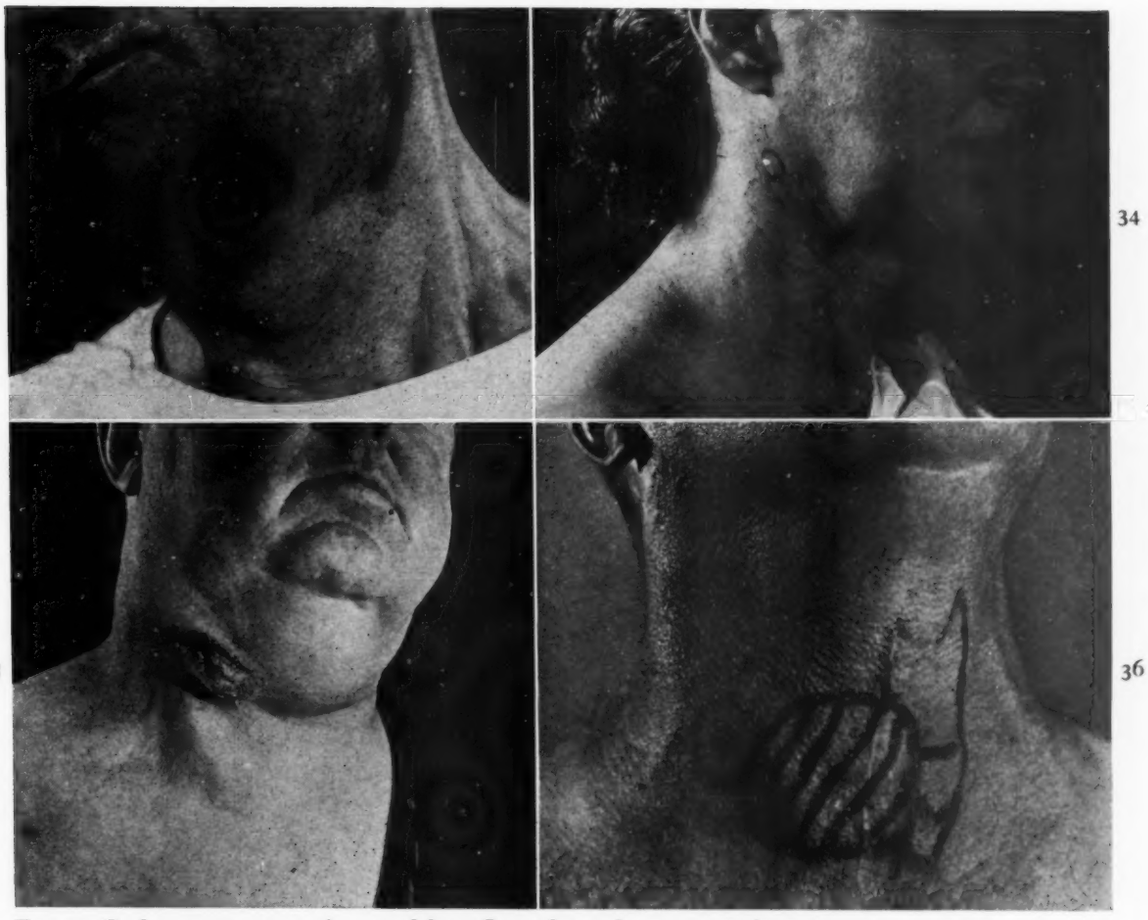


FIG. 33. Oral cancer; metastatic spread from floor of mouth; man, age sixty-one.

FIG. 34. Oral cancer; metastatic spread from primary near angle of jaw and in front of tonsillar pillar along gingival margin; woman, age forty-one.

FIG. 35. Oral cancer; metastatic spread from primary in posterior base of tongue; man, age seventy.

FIG. 36. Carcinoma, metastatic, from larynx; man, age sixty-seven.

nodule in a similar position in a forty-four year old man which proved to be lymphosarcoma.

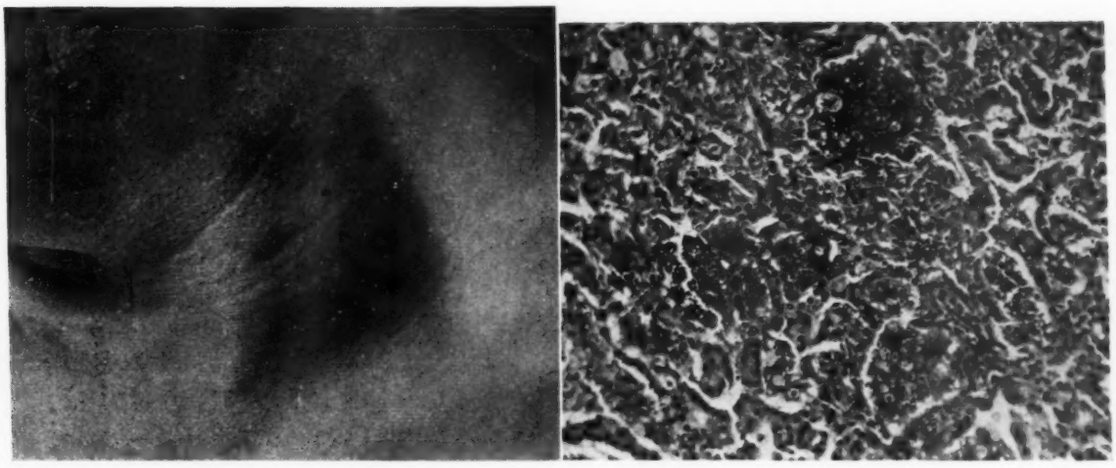
METASTATIC CARCINOMA

Perhaps numerically the largest number of neck masses that we have to deal with today (excluding goiter) are metastatic carcinomas from the oral cavity. Diagnostically we shall consider only one point pertinent to this discussion. By and large, these growths follow the lymph channels from the primary disease in the mouth. This, of course, is a simple and evident fact but, if kept in mind, a great deal of diagnostic confusion can be eliminated. Cancer of the lip goes directly to the nodes posterior to the submaxillary gland. (Fig. 31A.) It may also form large masses along the mandible, so-called "watermelons," often invading the bone. (Fig. 31B.) The exception is a primary growth in the mid-portion of the lip; from this site it goes to the submental nodes underneath the midline of the chin. Cancer primary in the side of the tongue proceeds first to the digastric nodes just below the submaxillary region. It may be confused with a submaxillary tumor or a salivary calculus. (Figs. 32A and B.) From the floor of the mouth cancer metastases may go to the digastric nodes but usually appear a trifle lower along the sternomastoid region. (Fig. 33.) If the primary is more posterior, toward the tonsillar region, the neck node first to appear lies immediately behind the angle of the jaw. (Fig. 34.) Growths arising in the base of the tongue commonly metastasize low

Fig. 37. Carcinoma, metastatic, from primary growth in nasopharynx; man, age seventy; large bulky lateral neck masses with often a small scarcely distinguishable primary. These have been confused with carotid body tumors and supposed branchial cyst carcinomas (latter probably do not exist).

Fig. 38. Carotid body tumor of long duration, typically fixed vertically and movable laterally and located exactly at the carotid bifurcation; woman, age sixty-seven.

Fig. 39. Virchow's node; primary growth was carcinoma of the stomach; man, age sixty-nine.



40A 40B

FIG. 40. A and B, neck metastasis from hypernephroma; man age fifty-four.

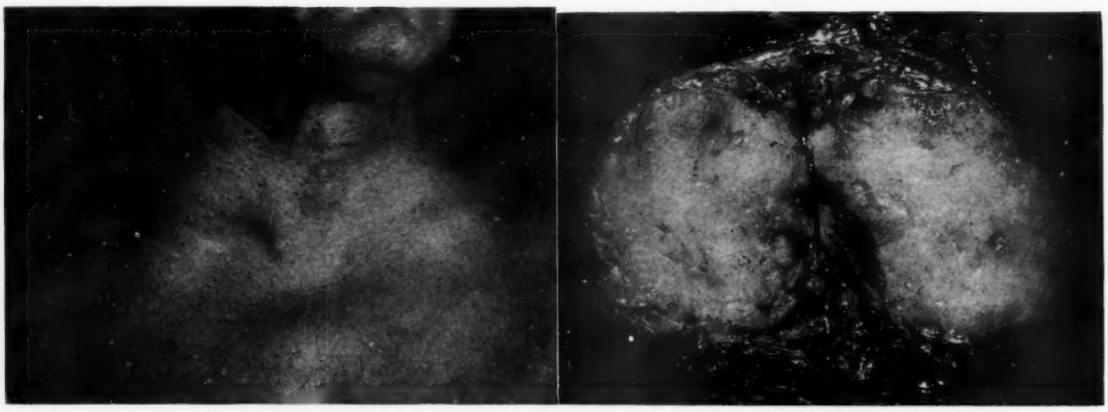
down along the sternomastoid muscle. (Fig. 35.) Cancer of the larynx may appear along the larynx and the trachea (Fig. 36), or more usually a bit higher and more lateral to the upper portion of the larynx below the hyoid region.

A clinical entity which Hayes Martin reported a few years ago is a massive bulky metastatic carcinoma in the lateral side of the neck which is secondary to a very small primary growth in the nasopharynx. (Fig. 37.) It is an example of tissue specificity wherein the primary growth remains very small and the secondary in another tissue attains tremendous size. The diagnostic significance is that the primary is often minute, may not be seen in careful nose and throat examinations for several months (sometimes it is entirely eradicated by x-ray therapy and can never be seen) and the neck masses frequently are bulky and often bilateral. They have been confused with carcinoma arising in branchial cysts which are in the same location. Martin believes that there is no such entity as carcinoma developing in the branchial cyst; it just does not occur. The condition is hopeless and all that can be done is to give palliation by x-ray therapy.

TUMORS OF THE CAROTID BODY

Tumors of the carotid body are not large, are relatively slow growing, are fixed directly at the bifurcation of the carotid with no up and down mobility and with a small amount of lateral mobility. They present a single, small, or moderate-sized, soft, fixed mass. Figure 38 shows a patient who was diagnosed preoperatively chiefly because of the location and slow growth. On exploration, it expanded diffusely over the entire crotch of the carotid artery. Nothing was done. Recent published reports during the past few years indicate that more of these have been resected. The danger, of course, is the frequent necessity of ligating the common carotid and the ensuing cerebral thrombosis (one-third of the cases). One can dissect these tumors and leave only the intima, thereby removing the adventitious layer of the carotid artery. However, there are differing opinions. If it is a very slow-growing tumor in an older person, the author believes it wisest to leave the tumor unless resection can be

FIG. 41. Metastasis in mandible and submaxillary triangle from primary in lung; man, age seventy-three.



42A 42B

FIG. 42. A, undifferentiated tumor right supraclavicular region. Biopsy: primary source uncertain: one opinion, from nasopharynx; another, from lung. Not thyroid in origin; patient did not respond to x-ray therapy; lower radical neck dissection; patient well after four years. B, gross specimen.

done with more ease than was demonstrated in this case.

NECK METASTASIS FROM INFRACLAVICULAR MALIGNANCY

Finally, one must remember that metastatic disease appears in the neck from primary sources below the clavicle. Virchow's node needs only to be remembered to be properly evaluated. Figure 39 shows a man who was referred for gastrectomy for a carcinoma of the stomach with a six month history of epigastric distress. The node made a total difference in the therapy. Carcinoma may appear in the neck from visceral sources other than the stomach. Figures 40A and B shows a metastatic focus from a hypernephroma. Occasionally, carcinoma metastatic from below the clavicle may appear in other locations in the neck than the usual supraclavicular region. Figure 41 shows a metastasis in the mandible and the contiguous neck from an oat cell carcinoma of the lung.*

Occasionally a tumor can be found which, although presumably metastatic, cannot be traced to any primary focus. Sometimes such a tumor can be removed with profit to the patient. (Fig. 42.)

* Castigliano and Rominger have recently reviewed the literature on Metastatic Malignancy of the Jaws (*Am. J. Surg.*, 87: 496, 1954) and reported seven cases. They found 176 cases reported up to 1953. Metastasis to the jaws may be the first evidence of the disease. The thyroid was the most frequent source. Almost 70 per cent were metastatic from the thyroid, breast, hypernephroma and the lung; thirteen of the 176 were primary in the lung.

SUMMARY

Anatomic location plays a large role in the accurate diagnosis of tumors of the neck. (1) Thyroglossal cysts may appear lateral to the midline. (2) Papillary adenocarcinoma of the thyroid (previously termed carcinoma primary in aberrant thyroid tissues) may appear anywhere in the midline from the nasopharynx to the mediastinum, including the trachea and the esophagus, and almost anywhere in the lateral neck regions. It frequently appears as isolated masses, either single or multiple, along the jugular chain or underneath the lateral or posterior border of the sternomastoid muscle. It may even appear laterally near the acromion and outer end of the clavicle. (3) Various simple benign conditions must always be thought of in diagnosing neck lesions; especially sebaceous cysts, lipomas, mild inflammatory conditions and tumors arising from superficial nerves, fascia, muscles, etc. (4) Lymphomas present diagnostic problems which often prevent an accurate solution until the microscopic section is obtained, and even then there may be confusion among the pathologic interpretations. (5) Oral metastatic carcinoma follows the lines of the lymphatic drainage in the neck. The diagnosis often rests upon the accurate interpretation of this lymphatic drainage.

Acknowledgment: We wish to thank Dr. Wm. L. Brosius, pathologist, Harper Hospital, for his interest and cooperation. Photography was done by Frank Russlander of the Harper Hospital.

Papillary Adenocystoma Lymphomatosum (Warthin's Tumor)

CORNELIUS E. SEDGWICK, M.D. AND ROBERT E. SASS, M.D., *Boston, Massachusetts*

From the Department of Surgery of the Lahey Clinic, Boston, Massachusetts.

IN 1895 Hildebrand[5] reported the first case of papillary cystadenoma lymphomatosum. Albrecht and Artz[1] in 1910, however, established this diagnosis as a distinct entity and are credited with giving it its present name. Warthin[10] reported two cases in 1929 and described the histopathology, and his name has occasionally been given to this specific neoplasm.[6,7] Other names include adenolymphoma,[2,4] epitheliolymphoid cyst, oncocytoma, branchioma, branchiogenic adenoma of the lymph nodes, orbital inclusion adenoma, cylindrocellular branchiogenic adenoma and branchiogenous cystadenolymphoma. The long and varied nomenclature stems from the inability of investigators to agree on the possible[2–4,6–9] origin of this neoplasm.

Some of the proposed theories of histogenesis are as follows: (1) aberrant anlagen of the eustachian tube; (2) heterotopia of pharyngeal entodermal anlage in lymph nodes near the parotid gland; (3) remnants of a branchial arch; (4) orbital inclusion cyst; (5) ectopic tonsil; (6) origin from a branchial cyst; (7) evaginations of the embryonal buccal endoderm; (8) undifferentiated salivary tissue; (9) thymic anlage; (10) salivary gland ducts; and (11) oncocytes, which are large cells with hyperchromic nuclei and finely granular eosinophilic cytoplasm found in the salivary duct, most often in elderly persons.

It is not the purpose of this paper to attempt to establish the histogenesis of this tumor. Suffice to say that the epithelium and lymphoid tissue seen in this tumor are not the same as those seen in adult life. They are the embryonal type of epithelium and lymphoid tissue seen in other developmental anomalies. In view of this it must be assumed that the tumor is a developmental anomaly of unknown origin.

This neoplasm is round, encapsulated and usually lobulated. It cuts with ease and is composed of a pink grayish or yellow trabeculated tissue. The cut surface may present small cystic areas containing brown fluid or it may be pulpy and have a papillary structure. Histologically, the stroma is composed of reticulum containing lymphocytes and well developed lymph follicles. Embedded in this stroma are epithelial cells arranged in a cystic or papillary pattern. The epithelium is arranged in one or two layers, beneath which is a row of basal cells on a basement membrane. The epithelial cells are tall, columnar and have a fine granular eosinophilic cytoplasm. The basophilic nuclei are located in the distal half of the cells. Cilia have been reported on the inner surface but are not a constant finding.

A series of twenty-one patients with papillary adenocystoma lymphomatosum seen at the Lahey Clinic during the past eight years was studied. It has been estimated that papillary adenocystoma lymphomatosum constitutes 5 to 6 per cent of all parotid tumors occurring in both sexes. In our series there were seventeen men and four women. It has been reported to occur from childhood to old age, the commonest occurrence being in the fifth to seventh decades of life. The average age of patients in this series was fifty-nine years.

Clinically, the patient usually gives a history of a painless mass at the angle of the jaw in the region of the parotid gland. Aside from this distribution, the neoplasm has reportedly been found in the submaxillary gland; lateral to the upper pole of the thyroid; the posterior auricular area; the anterior margin of the sternocleidomastoid muscle, and the bifurcation of the carotid artery. In this series four tumors were in the infra-auricular area and seventeen were below the angle of the jaw. The length of time the swelling had been present varied from several months to several years. The average duration in this series was four and one-half years, the shortest being one

and one-half weeks and the longest twenty years. In all of our cases the tumors were unilateral, fourteen being on the right side and seven on the left. In only one case was the mass discovered on routine physical examination; its presence was unknown to the patient at the time of the examination.

Occasionally the patient may complain of slight discomfort in the region of the tumor. In five of our cases there was slight pain to severe aching. However, the mass is usually discovered accidentally by the patient and because of lack of symptoms, several years may elapse before medical advice is sought.

On physical examination the tumor is distinct in outline and is not attached to the skin or deeper structures. It may vary from soft to firm on palpation. Tenderness is usually absent unless secondary infection is present. Frequently, from the clinical findings, if the mass is in the region of the parotid gland, it is impossible to distinguish this tumor from a mixed tumor of the parotid gland. The diagnosis is accurately established only after excision and pathologic examination of the specimen. It is usually wise to obtain a frozen section study at the time of excision to determine whether surgical extirpation is necessary.

The treatment of papillary adenocystoma lymphomatosum is surgical excision. When the tumor is in the region of the parotid gland, the same care is taken to preserve the facial nerve as in operations on the parotid gland for other types of tumors.

In a series of twenty-one patients with papillary adenocystoma lymphomatosum treated at the Lahey Clinic in the past eight years there has been one recurrence. In four patients excision had been attempted previously at other hospitals but these tumors have not recurred since their removal at this institution. In one of our cases the tumor mass appeared several months after the original excision. It was again excised and the patient's course has been followed for the past eight years with no evidence of recurrence. In this series of cases, facial paralysis or salivary fistulas have not occurred.

SUMMARY

Papillary adenocystoma lymphomatosum is an unusual tumor, probably of developmental histogenesis. It usually occurs in the region of the parotid gland. Clinically, the tumor cannot be distinguished from other tumors affecting the parotid gland, particularly mixed tumors. Surgical treatment is total excision of the tumor and recurrence is rare.

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Surgical Management of Complete and Incomplete Bony Atresia of the Posterior Nares (Choanae)

WITH DETAILED DESCRIPTION OF AN IMPROVED TECHNIC

HENRY H. BEINFIELD, M.D., *Brooklyn, New York*

From the Ear, Nose and Throat Department of the Polhemus Clinic, the Long Island College Hospital; and the Department of Otolaryngology, State University of New York, College of Medicine at New York City. Presented at a meeting of The American Otorhinologic Society for Plastic Surgery, Inc., on March 27, 1954 at the Hotel Statler, Boston, Massachusetts.

CONGENITAL atresia of the posterior nares is not seen too often; yet judging by the number of papers on this subject appearing in the literature, this condition is now being recognized more frequently than heretofore. It therefore becomes a timely subject to discuss and especially to describe a logical surgical technic for its correction.

It might seem strange to present a paper of this kind before a society on plastic surgery since the subject does not deal directly with a purely cosmetic result. The treatment of atresia of the posterior nares nevertheless is just as much a part of a program on plastic surgery as is a harelip or cleft palate, because the principles of plastic surgery are employed for its correction. The primary principle involved is the creation of two raw mucous membrane surfaces facing each other which heal together to cover a newly created bare bony rim in the posterior nares, thereby preventing its closure. This is an entirely different kind of a situation in which plastic surgery is applied and used to good advantage.

Since 1853, when the first case was seen, up to the present time, many and varied technics have been tried in order to obtain a permanent natural opening in the posterior nares, but without success. The surgery at the beginning was very simple but then it became more technical, with no better results. This fact is significant in demonstrating that no uniform procedure has yet been established. The following is a résumé of the various trial and error types of surgery and instruments that have been used for the purpose: The first attempt at making an opening in the posterior nares was made with a trocar; this was then followed by the use of the galvanocautery, chemical cauterization, a hand drill, chisel and curet. As the technic improved, a septal flap was created and the posterior end of the vomer was removed with a rongeur. Later a submucous resection was performed with the removal of the posterior third of the septum. A transpalatine approach was described and the vomer alone was removed. A split skin graft and then a full-thickness graft was wrapped around a catheter and inserted into the opening. A sphenoid punch was used to remove the posterior edge of the septum at the time an opening was created. Electrocoagulation was employed. The perpendicular plate of the ethmoid bone was removed after perforation of the atresia with a chisel. The mucous membrane over the anterior surface of the atresia was elevated after a submucous resection was performed, and its elevation was followed by the removal of ½ inch of the membranous septum. The posterior part of the vomer was removed and the mucous membrane split, through which a No. 18 catheter was inserted. Submucous resection was performed with the removal of the vomer and posterior third of the septal soft tissue. An approach by way of the antrum was tried, and so forth.

By following the evolution of these methods it can be noted that attempts to create a permanent opening in the posterior nares were finally abandoned. It soon became apparent that after drilling, puncturing, etc., the opening in the posterior nares would eventually close. After such disappointments attempts were

FIG. 1. Patient operated upon for the correction of a left incomplete bony atresia. Note the anomalous flatness of the malar eminences, producing a strange facial appearance.

made to by-pass the atresia of the posterior nares and to connect the obstructed side with the unobstructed side by removing the vomer and posterior septum. Others more properly tried to graft the opening with skin but were unable to bring the graft into the opening in the back of the nose. The idea was a good one but not practical. After a proper evaluation of the foregoing technics we must admit that all of them were more or less destructive and none succeeded in producing a permanent, natural-sized opening. The reason these methods failed is that the approach and anatomy were not understood or taken into consideration. The tissues which are present and could be utilized for the elimination of the atresia were destroyed. The technic which I shall describe does not destroy any of the tissues. Instead, these tissues are preserved and fashioned in such a manner as to be surgically, anatomically and physiologically correct, with a permanent opening as the final result.[1]

Embryology. Embryologically, atresia of the posterior nares is believed to be due to the persistence of the bucconasal membranes with a mesodermic layer between the two membranes. The mesodermic layer later develops to form the bony portion of the atresia. This should mean that the atresia in infants is membranous, but occasionally it is bony as reported by others and myself.

Anatomy. Anatomically, atresia of the posterior nares is a congenital anomaly, situated at the junction of the hard and soft palate, completely closing off the posterior choana on that side of the nose. It may be unilateral or bilateral. There are two definite types, namely, complete bony atresia and incomplete bony atresia. A third type may exist, which I term partial atresia. Complete bony atresia can be defined as one in which the posterior choana is completely closed by bone, with the bone entirely covered by the nasal mucous membrane in front and by the pharyngeal mucous membrane behind. Incomplete bony atresia is one in which the atresia, although complete in form, is only partly bony, the remainder of the closure being membranous. The membranous portion is very thick, apparently due to a fusion of both the nasal and pharyngeal mucous membranes. Where the membranous portion meets the bony portion, the membranes split or separate into a nasal and pharyngeal membrane to envelop the bone where it exists. Partial atresia would be one in which the closure has one or more openings in it, through which a small amount of air might pass. An understanding of this anatomy is very important to appreciate the rationale of my technic.

Anomalies. It is not unusual to find other anomalies associated with atresia of the posterior nares. One of my cases presented a facial asymmetry, the other an absence of both malar eminences. (Fig. 1.) Other men reported a double tragus on each ear, a coloboma of the iris and cleft palate. A hereditary

relationship in some of these cases has also been reported.[1,2]

Symptoms. These patients complain chiefly of complete nasal obstruction on one side of the nose, accompanied by a thick mucoid discharge. On examination of the nose the inferior turbinate on the obstructed side is pale and edematous, with marked redundancy of the mucous membrane, almost polypoid in nature. Thick glairy mucus is found in the inferior meatus. These patients are occasionally operated upon for the correction of a deviated septum to relieve nasal obstruction, only to find, to the surprise of the surgeon, that atresia of the posterior nares exists.

Diagnosis. The diagnosis is comparatively simple. Atresia should be suspected if the nasal obstruction is complete. In the older child the posterior nares can be routinely examined, with the finger in the nasopharynx when removing adenoids in every tonsil and adenoid case. In this manner an occasional case of atresia of the posterior nares may be found to account for a history of unrecognized nasal obstruction. Confirmation of the diagnosis can be made with a probe or by the inability to pass a catheter or dye into the nasopharynx through the nose, by postnasal examination with a mirror or the finger, and by x-ray studies with lipiodol. For greater contrast by x-ray it is advisable to shrink the mucous membrane before instilling the lipiodol. There is no method of making an anatomic diagnosis, either clinically or by x-ray, to determine in advance whether the atresia is completely or incompletely bony.

OPERATIVE TECHNIC

The initial incision of the operation is made as far forward as possible because the innermost depth of the nose cannot be approached in any other way. No technic that I know of has ever made use of the floor of the nose as a means of access to the posterior nares. These two points are the basis for the atresia operation to be described.

This operation for the correction of bony atresia of the posterior nares is very simple to perform. Its use is primarily limited to the older child or adult; a different technic is used for the infant.[1] A good headlight is necessary for proper illumination. Any mucus in the nose is removed before beginning the surface anesthesia. Pledgets of cotton soaked with 2 per cent pontocaine or equal parts of 10 per cent cocaine and 1:1000 adrenalin are then placed in the nose, one pledget being placed against the septum, another on the floor of the nose and a third against the inferior turbinate. These are allowed to remain in place for a few minutes and are then removed. The nasal mucosa is then well infiltrated with 1½ per cent monocaine with 1:30,000 epinephrine, in the same manner as for submucous resection of the septum. The injection is best started on the floor of the nose anteriorly, carrying it to the nasoantral wall and then onto the lower half of the septum. The posterior part of the nose is also thoroughly infiltrated. If the infiltration is made deeply, elevation of the mucous membrane will be greatly facilitated. After the anesthesia has been completed, it is very important to fracture the inferior turbinate upward so that its inferior border assumes the position of a superior border. (Fig. 2A and B.) This procedure increases the working area in the lower half of the nose by taking advantage of the space normally occupied by the inferior turbinate. A U-shaped incision is now made, starting it along the floor of the nose just behind the anterior edge of the maxilla, carrying it down to the bone and then up the nasoantral wall to the attachment of the inferior turbinate on one side and onto to the lower half of the septum on the other side. (Fig. 3.) The mucoperiosteum elevates far more easily when the incision is started on the floor of the nose. Here it readily finds the line of cleavage for the elevation of the mucosa on the septum and the lateral wall. A sharp septal periosteal elevator is used for the elevation, closely hugging the bone while extending the elevation to the back of the nose until the bony atresia is reached. The danger of perforation is practically nil. There is little or no bleeding during the operation. After the nasal mucous membrane has been elevated, a long-billed Killian nasal speculum is introduced between the elevated nasal mucosa and the bone of the floor of the nose, vomer and nasoantral wall. By widely spreading the Killian speculum, the bony portion of the atresia comes into view and is now visualized in its entirety. (Fig. 4.) If the atresia is completely bony, the entire posterior choana will be seen to be closed only by bone. However, if the atresia is incompletely bony, the amount of bone present to

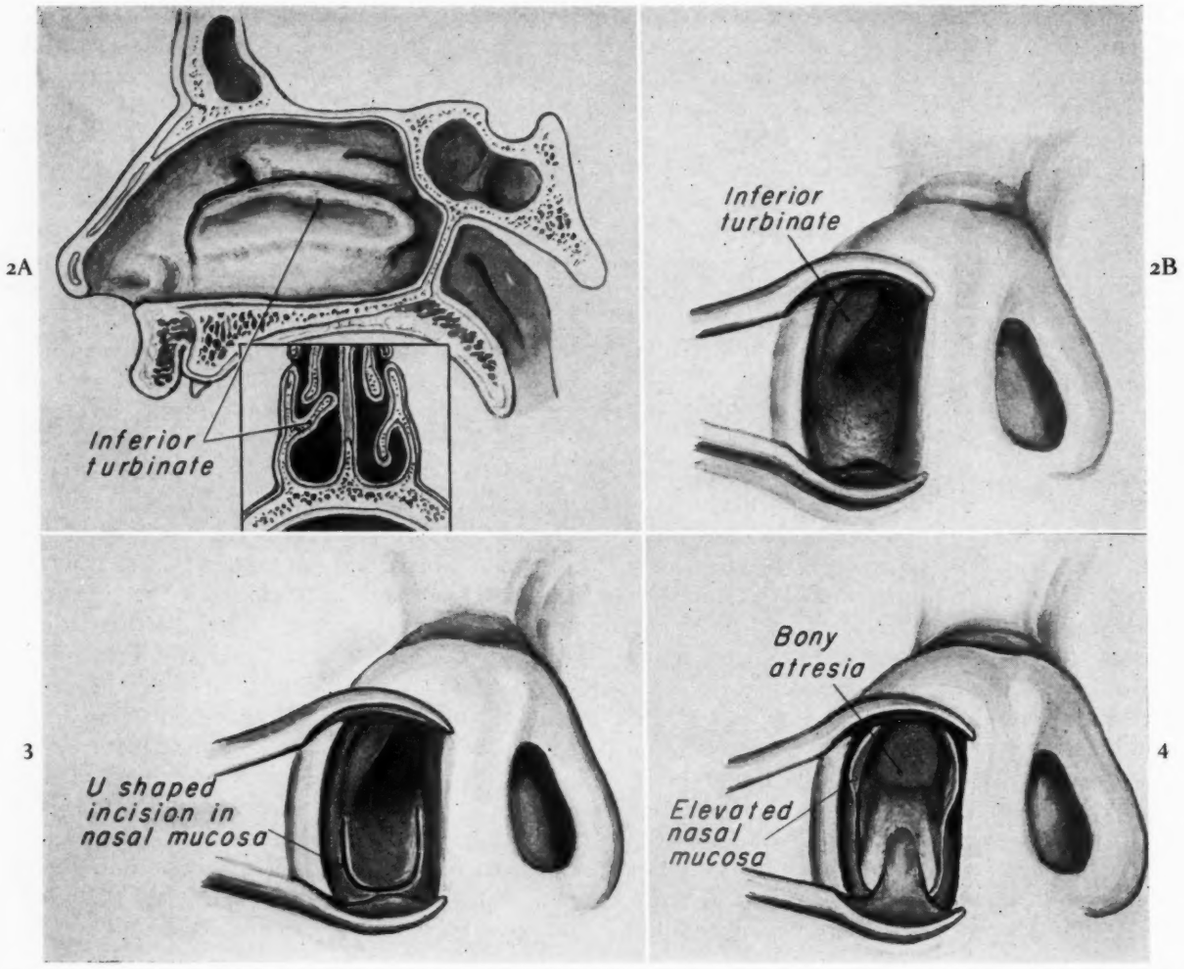

FIG. 2. A, inferior turbinate is fractured upward, so that its inferior border assumes the position of a superior border. This procedure increases the working area in the lower half of the nose by taking advantage of the space in the inferior meatus (sagittal view insert). B, same as Figure 2A, as seen through nasal speculum.

FIG. 3. The U-shaped incision is started on the floor of the nose, just behind the anterior edge of the maxilla, bringing it down to the bone. From here it is carried up the nasoantral wall to the attachment of the inferior turbinate on one side and halfway up the septum on the other side.

FIG. 4. The nasal mucous membrane has been elevated from the floor of the nose, nasoantral wall and septum exposing the complete bony atresia. The elevated nasal mucous membrane is held above the upper blade of the Killian nasal speculum.

be exposed is less; and its exposure therefore is limited by the remaining membranous portion of the atresia which closes off the rest of the choana. (Fig. 5.) In complete bony atresia the bone is freed from its attachment with a No. 10 gouge on a level with the floor of the posterior choana, the septal side, roof and lateral wall. (Fig. 6.) By tapping the gouge lightly until the sense of resistance is lost, the danger of perforation will be prevented. The same procedure is followed for incomplete bony atresia except that there is less bone present to free. After the bony portion is separated from its attachment, it is easily peeled off the pharyngeal mucous membrane with a periosteal elevator or bayonet forceps. At this stage of the operation a new, bare bony rim is created which will eventually be the permanent natural opening. The posterior choana is now thoroughly clear of any bone but is still completely closed by the pharyngeal mucous membrane which is present. To produce the final opening in the complete bony atresia type, the pharyngeal and nasal mucous membranes are each treated separately. This is accomplished by first

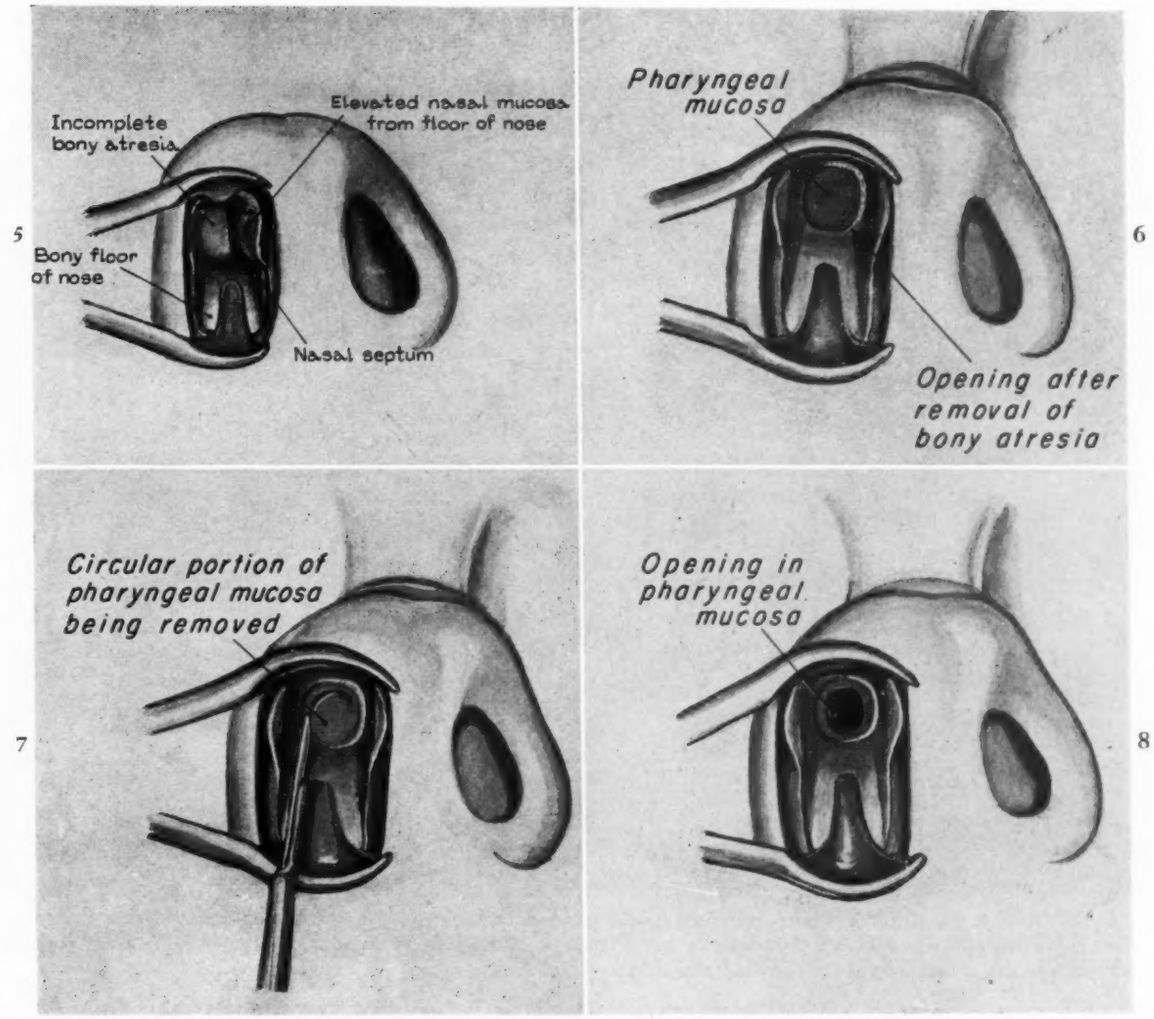

FIG. 5. That part of the nasal mucous membrane covering the incompletely bony portion of the atresia has been elevated exposing the bony portion of the atresia. The rest of the nasal mucous membrane has been elevated from the floor of the nose, etc., and demonstrates where it fuses with the pharyngeal mucous membrane to form the membranous portion of the atresia. The membranous portion of the atresia is behind the elevated nasal mucosa mesially and is not seen.

FIG. 6. The complete bony atresia has been separated with a No. 10 gouge from its attachment on a level with the floor of the nose, the septal side and nasoantral wall of the posterior choana. This figure shows that the bony atresia has been removed from the pharyngeal mucous membrane, demonstrating the bare bony rim of the permanent newly created natural opening with the pharyngeal mucous membrane still intact.

FIG. 7. With a No. 11 Bard Parker knife a circular portion of the pharyngeal mucous membrane is being removed, leaving a small rim of its mucosa overhanging the newly created bony rim.

FIG. 8. The circular portion of the pharyngeal mucous membrane has been removed as the first step in making an opening in the posterior nares. The nasopharynx is seen through the opening. The nasal mucosa is still held elevated by the Killian speculum.

removing a circular portion of the pharyngeal mucous membrane with a No. 11 Bard Parker blade (Fig. 7), leaving a small rim of mucosa around its circumference overhanging the newly created bare bony rim. (Fig. 8.) When doing this, care is taken to protect the posterior pharyngeal wall from injury with the knife by keeping the finger in the nasopharynx against the opening closed by the pharyngeal mucosa. The Killian speculum is now removed and the previously elevated nasal mucosa is returned to its former place on the septum, floor of the

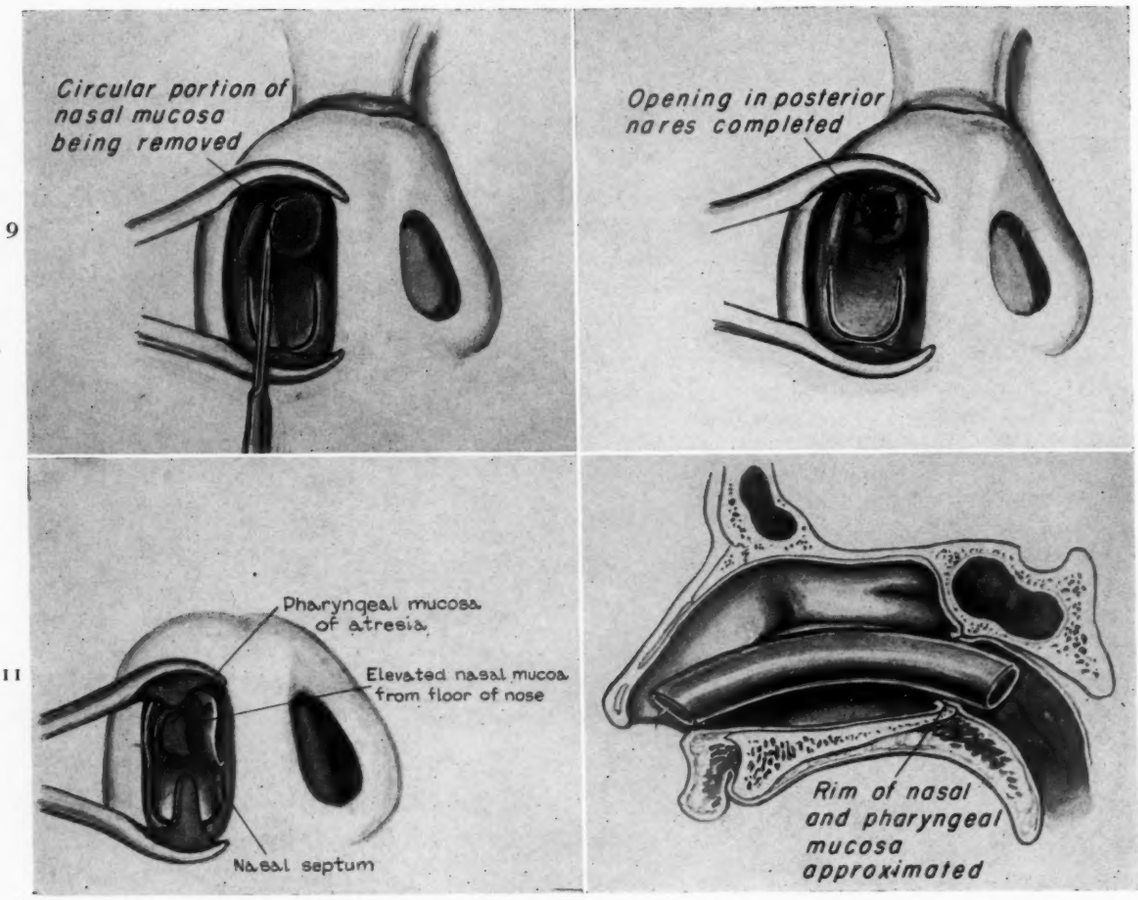

FIG. 9. The Killian speculum has been removed and the previously elevated nasal mucosa returned to its former position. This procedure now closes the posterior nares from the nasal side. A circular portion of the nasal mucosa of the posterior nares, similar to that of the pharyngeal mucosa, is also removed.

FIG. 10. The posterior nares is now completely open. Stellate incisions are made through the nasal and pharyngeal mucous membranes which overhang the newly created bare bony opening. The raw surfaces of these two membranes face each other and heal together around the bare bony rim, thereby preventing it from closing.

FIG. 11. In the incomplete bony atresia the bony portion has been removed, exposing the pharyngeal mucous membrane which joins with the nasal mucous membrane at the junction where the bone is absent to form the membranous portion of the atresia mesially. The membranous portion is behind the elevated nasal mucosa.

FIG. 12. The rubber tubing, which is twice the size of the opening of the anterior nares, is passed into the nose by way of the nasopharynx, approximating the nasal and pharyngeal mucous membranes which overhang and cover the newly created bare bony opening. The tubing is removed after five days to one week. The inferior turbinate returns to its previous position after the tubing has been removed.

nose and nasoantral wall. The replacement of the nasal mucosa again closes the posterior nares from the nasal side. A circular portion of the nasal mucosa is also removed, similar to that removed from the pharyngeal mucous membrane. (Fig. 9.) This last procedure now completely opens the posterior choana, leaving two circular strips of mucous membrane overhanging the newly created bony rim, one on each side of it with their raw surfaces facing each other. (Fig. 10.) When these membranes heal together, this newly created natural opening becomes completely covered by its own nasal and pharyngeal mucous membranes, thereby preventing it from closing. Either membrane alone could also accomplish the same purpose. In incomplete bony atresia the membranes are practically one and are handled as such. (Fig. 11.) In this case the circular portion of membrane is removed from the entire thickness of the membranous atresia after the nasal mucous membrane has been re-

placed. The remaining rim of mucous membrane around the newly created opening can be incised in a stellate fashion or it can be left alone. Using the technic of placing a postnasal pack, a piece of rubber tubing is passed into the nose by way of the nasopharynx to insure that the raw surfaces of the two mucous membranes are approximated around the bare bony rim. (Fig. 12.) Since the posterior nares is two and a half times as large as the anterior nares, the diameter of the tubing used should be about twice the size of the opening of the anterior nares. The presence of the rubber tubing in the nose does several things. It exerts pressure against the replaced elevated nasal mucous membrane on the floor of the nose, nasoantral wall and septum, eliminating the possibility of any dead space; it prevents formation of a hematoma and minimizes any postoperative bleeding. In infants if the atresia is membranous a crucial incision can be made in the posterior nares and a rubber catheter placed through it. The rubber tubing is removed from five days to one week after operation, with no further treatment necessary. The previously fractured inferior turbinate returns to its normal position after the tubing is removed. A submucous resection can be performed at the same time, if necessary.

COMMENTS

The operation described is simple, effective and non-destructive. It is surgically, anatomically and physiologically correct in every way, creating as a final result a permanently patent, natural-sized opening.

It might be added that the presence of bilateral atresia of the posterior nares is frequently responsible for asphyxia in infants at birth.[1] The unexplained cases of suffocation in infants up to five months of age who are found dead in their cribs or carriages may be due to unilateral atresia when no other definite pathologic cause can be found at autopsy.[2]

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The Continuing Problem of Infection and Gangrene in the Diabetic Extremity

CHARLES W. MCLAUGHLIN, JR. M.D. AND CHARLES F. HEIDER, JR., M.D., *Omaha, Nebraska*

From the Department of Surgery, University of Nebraska College of Medicine, Omaha, Nebraska.

INFECTION and gangrene in the diabetic extremity has always been a distressing and discouraging problem. From 1932 to 1953, 135 such cases were seen at the University of Nebraska Hospital. They represented 7 per cent of a total of 1,912 diabetic admissions in the same period. One of us (C. W. M.) has previously reported on part of this series.[2] We are presenting this problem as it has existed at the University Hospital in the past twenty-two years, giving our experience with its treatment. The majority of these patients come to us from rural communities, farms and ranches. Many were first seen by their local physician when infection and gangrene were well established. Regular diabetic care at a medical clinic was an exception. These factors must be kept in mind in analyzing our results in treatment.

TABLE I
KNOWN DURATION OF DIABETES

Time	Cases	Percentage
Unknown on admission	17	12.5
1 mo. to 1 yr	13	10
1 to 2 yr	12	9
2 to 5 yr	21	15.5
5 to 10 yr	29	21
Over 10 yr	43	32

There was some predominance of males in this group (75:60), although the average age of each sex was the same, sixty-one years. This does not imply that this is a condition found only in the older arteriosclerotic age group. Our youngest patient was twenty-seven years of age, and the oldest eighty-one.

Many of the patients were unknown diabetics on admission, particularly during the early years of the review. (Table I.) This has not occurred during the past five years. The majority of the patients had been known diabetics for at least five years.

The type of medical treatment received before admission with gangrene is listed in Table II. Almost one-fourth of the patients in this series had received no treatment before admission. In recent years we have seen very few patients who have not had some sort of treatment, undoubtedly due to better economic conditions in our state. Nevertheless, over one-half had received either poor or no treatment. It is interesting to note the very small percentage with infection and gangrene who had been on good diabetic management. Certainly ignorance and indifference toward this condition are obstacles difficult to overcome. Possibly a more intensive educational program would help. Teaching patients the simple methods of testing urine, regulating diet and administrating insulin as well as proper care of their feet would unquestionably be of prophylactic value and would reduce the incidence of gangrene.

TABLE II
PREVIOUS TREATMENT FOR DIABETES

Treatment	Cases	Percentage
None	31	23
Poor	43	32
Fair	49	36
Good	12	9

Prophylactic foot care is very important in prevention of gangrene in the diabetic. As illustrated in Table III, 40 per cent had some sort of precipitating factor for the development of infection and gangrene. Burns from hot water bottles, blisters from tight shoes, frostbite and injudicious paring of corns were the more common precipitating factors.

During the first years from which this series was gathered (1932 to 1942) extensive wet gangrene of the toes and foot often extending into the leg was the most common type of lesion encountered. These patients with toxemia, acidosis and uncontrollable diabetes and with the constant hazard of gas bacillus infection presented a very grave surgical emergency. (Table IV.) In the years following the advent of antibiotics these cases fortunately have been in the minority.

TABLE III
PRECIPITATING FACTORS PRECEDING ONSET OF GANGRENE

Factors	Cases	Percentage
Unknown	80	60
Trauma	16	12
Paring corns	10	7
Frostbite	9	6.5
Blisters	8	6
Ingrown toenails	2	1.5

TABLE IV
EXTENT OF DISEASE ON ADMISSION
(153 Hospital Admissions)

Disease	Cases	Percentage
Penetrating ulcers of feet	48	31
Dry gangrene of toes	30	20
Wet gangrene of toes	27	17.7
Wet gangrene (extensive)	46	30
Dry gangrene (extensive)	2	1.3

The relationship between arteriosclerosis and diabetes is now well established. The arteriosclerotic process continues to advance in spite of therapy, with the result that the diabetic patients' vessels usually are ten years older than the patient. Less importance has been attached to the presence of calcification in the extremities by x-ray in recent years. Apparently it has been assumed that calcification would be present. This assumption was correct in over three-fourths of the films that were taken. Osteomyelitis of the phalanges was also present in one-fourth of the films taken of the feet.

It is extremely difficult to standardize the treatment of diabetic gangrene in the general hospital. Each case presents a slightly different problem. The severity of the diabetes, adequacy of previous control of diabetes, and extent and severity of gangrene are but a few of the problems encountered. These problems should be managed by a small group of interested medical men and surgeons with close cooperation. This was not the case in our institution. Divided responsibility inevitably will result in a higher mortality and longer periods of hospitalization.

TABLE V
TREATMENT OF DIABETIC PATIENTS WITH ULCERS, INFECTION OR GANGRENE OF EXTREMITIES

Treatment	Cases (%)	Mortality (%)
1. Conservative	54 (35)	6 (11)
2. Conservative surgery		
a) Incision and drainage	11 (7)	0 (0)
b) Toe amputation	17 (11)	0
3. Primary major amputation	50 (33)	10 (20)
4. Primary conservative surgery with subsequent major amputation	17 (11)	4 (24)
5. Primary major amputation with reamputation	4 (3)	2 (50)
6. Lumbar sympathectomy (5 cases were bilateral)	13 (8.5)	1 (8)

Conservative treatment alone or combined with conservative surgery was instituted in over half of the whole series, with a constant mortality of around 10 per cent. With the progressive lowering of mortality rate (less than 10 per cent in past ten years) from major amputation more patients have been treated with more radical surgery. Better anesthetic methods, antibiotics and improved methods for managing diabetics has made this possible. Experience has shown that many of the patients treated by conservative methods, either surgical or medical, eventually required major amputations after a long period of hospitalization.

The importance of the proper site of election for amputation is brought out in Table V by the high mortality rate associated with reamputation.

Recently (1949–1953) lumbar sympathectomies have been performed as an additional therapeutic aid. Our experience certainly is limited, with a total of only thirteen cases.

Lumbar sympathectomy has no place in treating the severe diabetic with rapidly progressing degenerative disease. It should not be performed with the illusion that it will prevent major amputation, particularly in the absence of peripheral pulsations. However, in the occasional case it may influence the site of amputation. We believe that lumbar sympathectomy may have a more important place in prophylactic treatment. It is common knowledge that after one limb is amputated for diabetic gangrene, frequently it is a relatively short period of time before the other limb becomes gangrenous. For this reason we believe that it is worth while to perform a bilateral lumbar sympathectomy or at least a contralateral sympathectomy to the side of the gangrene as a prophylactic measure. This is important particularly if good peripheral pulses are palpable on the side opposite the gangrenous limb.

TABLE VI
TYPES OF MAJOR AMPUTATIONS EMPLOYED

Type	Percentage
Circular mid-thigh, drained	38
Circular mid-thigh, no drainage	17
Callander, no drainage	15
Callander, drained	17
Flap amputation below knee	10
Supracondylar guillotine	3

A mid-thigh amputation with short anterior and posterior flaps was the most common procedure employed in the whole series. (Table VI.) This also was true in a previous review made by one of us (C. W. M.) of cases in the 1932–1942 period. A rather marked change to the Callander amputation followed this period but recently the trend has been back to the mid-thigh amputation. This preference has been because of the lower percentage of wound infections, the extent of disease present and the relatively short operative time required. Rarely do these patients improve sufficiently to wear an artificial limb.

Amputation wounds are slower in healing and more prone to infection in the diabetic patient. In the early part of the series over 60 per cent of the wounds became infected following amputation, while recently this figure is less than 20 per cent. This has been attributed to use of wide spectrum antibiotics, meticulous surgical technic and proper selection of site of amputation, as well as better postoperative control of diabetes.

Diabetic gangrene is an expensive disease as proved by hospital records. Since the majority of these patients are aged and without funds, their care becomes the responsibility of charity or the taxpayer. The average number of hospital days for our entire series was forty-six; of those who survived, forty-seven days. Some progress has been made in decreasing the hospital stay in recent years but it is minimal. (Table VII.)

TABLE VII
DAYS OF HOSPITALIZATION

Average for entire group	46 days
Average for fatal cases	41 days
Average for non-fatal cases	47 days
Average for fatal cases before surgery	17 days
Average life of fatal cases after surgery	23 days

Further analysis of our series discloses that those patients who succumbed following operation were in the hospital for an average of seventeen days before surgery was undertaken. In certain cases it thus is apparent that there was entirely too long a period of conservative medical treatment before surgical consultation was requested. The diabetic patient with wet gangrene or spreading infection will not and cannot respond to medical treatment until the infection is overcome by surgical means. Conservatism on the part of the medical attendant can be quite as disastrous as the same tendency in the consultant surgeon.

The hospital mortality in this entire series was 16.3 per cent. This re-emphasizes the seriousness of the problem. This high mortality rate is discouraging but not hopeless, since it has been reduced from 22.6 per cent in the early part of the series to 10.2 per cent in the past few years. Sepsis associated with pneumonia was the cause of death in over one-third of the cases. Gas bacillus infection accounted for some deaths in the early part of the series but has not been a problem since the employment of antibiotics. Cerebral vascular accidents, cardiac failure and renal disease continue to take their toll.

CONCLUSIONS

1. A group of 135 cases of diabetic gangrene occurring in a series of 1,912 cases of diabetes is reviewed.

2. Good medical management of the primary

disease combined with meticulous care of the extremities is the most important factor in reducing the incidence of diabetic gangrene.

3. Close cooperation between experienced and interested internists and surgeons is needed to reduce mortality and length of hospitalization of these unfortunate patients.

4. Prompt radical surgery has reduced mortality and length of hospitalization.

5. Lumbar sympathectomy may be of value in selected cases.

6. The continuing high mortality rate and long periods of hospitalization have established diabetic gangrene as a distressing and expensive disease.

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Intravenous alcohol should be given to postoperative patients with glucose or a similar preparation, according to G. Grundmann, because "alcohol burns in the fire of carbohydrates." Furthermore, alcohol and glucose together have an antiketogenic effect and thus prevent postoperative acidosis. The author found that this therapy is sedative, analgesic and nutritive in effect, and often reduces the amount of postoperative morphine needed to keep the patient comfortable. He gives ½ L. intravenously in twenty-four hours (glucose 100.0, alcohol 35.0, distilled water ad 500.0) and thus the patient receives about 2,000 calories daily. The solution is given at the rate of 50 or 60 drops per minute. Unfortunately the vein may sclerose so a different side should be chosen for the infusion each day. The author prefers to use the distal veins rather than the medial cubital one. (*Richard A. Leonardo, M.D.*)

Joint Stabilization

AN EXPERIMENTAL, HISTOLOGIC STUDY WITH COMMENTS ON THE CLINICAL APPLICATION IN LIGAMENT PROLIFERATION

GEORGE S. HACKETT, M.D. AND DONALD G. HENDERSON, M.D., *Canton, Ohio*

From the Departments of Surgery and Pathology, Mercy and Aultman Hospitals, Canton, Ohio.

THIS twelve-month laboratory study of the histologic effects of a proliferating solution injected within the tendons of animals was undertaken to investigate the modus operandi and the clinical results which have been obtained during the past fifteen years in the strengthening of relaxed ligaments which re-established joint stabilization in man as reported by Hackett.

NEED FOR SCIENTIFIC DATA

Although the authors were satisfied that the clinical results of proliferation in strengthening relaxed ligaments to stabilize joints were especially satisfactory, it became increasingly evident that some physicians were skeptical of the method. It therefore seemed advisable to present evidence of the manner in which the clinical results were accomplished. Perhaps some skepticism was justified because the attempt to cure hernias by proliferation was disappointing. However, the treatment of varicose veins and some other conditions have been highly successful in competent hands. Also no previous scientific work had been done which demonstrated the volume of strong fibrous tissue which could be generated by the introduction of a proliferant within the joint ligaments, although the experiments had been made on some other tissue which revealed the production of permanent strong fibrous tissue.

Proliferation treatment of ligaments was initiated fifteen years ago when it became evident to one of the authors that much low back disability was due to relaxation of the joint ligaments as revealed by Mengert, Meisenbach and Magnuson. Proliferation promised the greatest reward in stabilizing the joint by promoting additional strength to the ligaments.

HISTORY OF SCIENTIFIC TISSUE PROLIFERATION

The scientific rationale for the injection of a proliferant was first established by Warren in 1881 when he reported the production of fibrous tissue which acquired strength in ten to twelve days. In 1929 Hall and Fraser using guinea pigs, monkeys and dogs demonstrated the vigorous new growth of endothelial and connective tissue cells following the injection of a proliferant.

In 1935 Rice and in 1936 Rice and Mattson reported the histologic results of sylnasol when injected in human tissue after periods from fifteen hours to forty-two days. Tissue sections were made from twenty-five patients. In three days fibroblasts appeared. In five days polymorphonuclear cells had largely disappeared. At fourteen days fibrous tissue was in dense bundles, and in forty-two days fibrous tissue was in *very* dense bundles.

In 1936 Harris, White and Biskind demonstrated microscopic illustrations of the fibrous tissue formation from two proliferants.

In 1938 Maniol using rats reported that in eight weeks firm scar tissue resulted without any undesirable histologic changes.

This is the first report showing the quantitative results of proliferating solution as well as the histologic results, and carried out over a sufficient length of time to demonstrate that the new fibrous tissue becomes permanent and that there are no degenerative sequelae.

ANIMAL EXPERIMENTATION

Although the clinical treatment has been applied to ligaments, the experimental work has been done on tendons because there are no ligaments in the smaller animals which are of sufficient size and accessibility to carry out the experiments. Ligaments and tendons are composed of non-elastic bands of similar white fibrous tissue, and both are attached to bones.

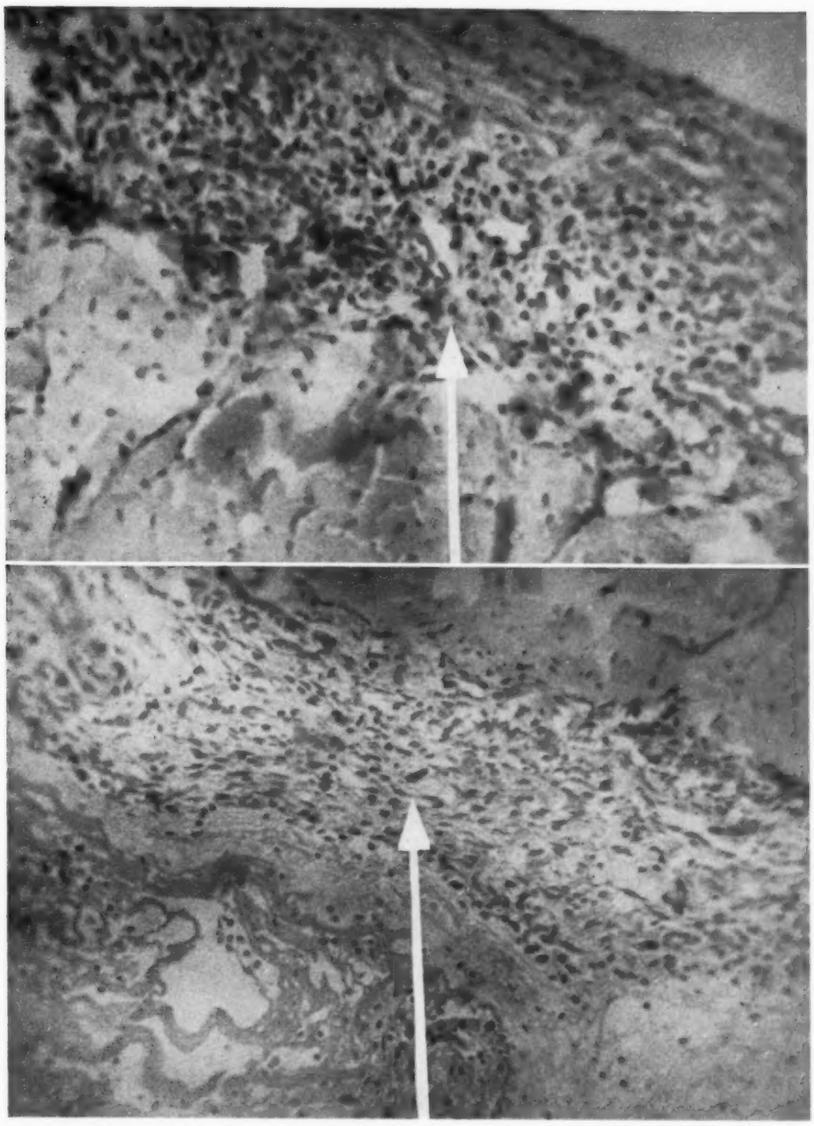


FIG. 1. Arrow points to moderate infiltration of lymphocytes forty-eight hours after injection of proliferating solution. Note absence of necrosis in surrounding tissue.

FIG. 2. Beginning fibroplastic organization present in adjacent tissues. Arrow points to capillary proliferation with moderate infiltration of lymphocytes. Two weeks after injection.

For this investigation the rabbit was selected. The experiments were made on the gastrocnemius and superficial flexor tendons where they are conjoined to the tuber calcanei of the tibial tarsal bone, analogous to the Achilles tendon in man.

The technic was similar to that used clinically. A preliminary injection of local anesthesia was infiltrated throughout the fibrous strands of the tendon as the needle was inserted. While the needle remained in place, the syringe was replaced by one containing the proliferating solution which was then injected along the course of the needle as it was withdrawn. The corresponding tendon on the other leg was not injected and was used as a control.

Microscopic views of sections represent effect of proliferating action at periods from one day to one year. (Figs. 1 to 4.) Ten rabbits were used in the series. The histologic examinations and photography were done in the pathology department of Aultman Hospital.

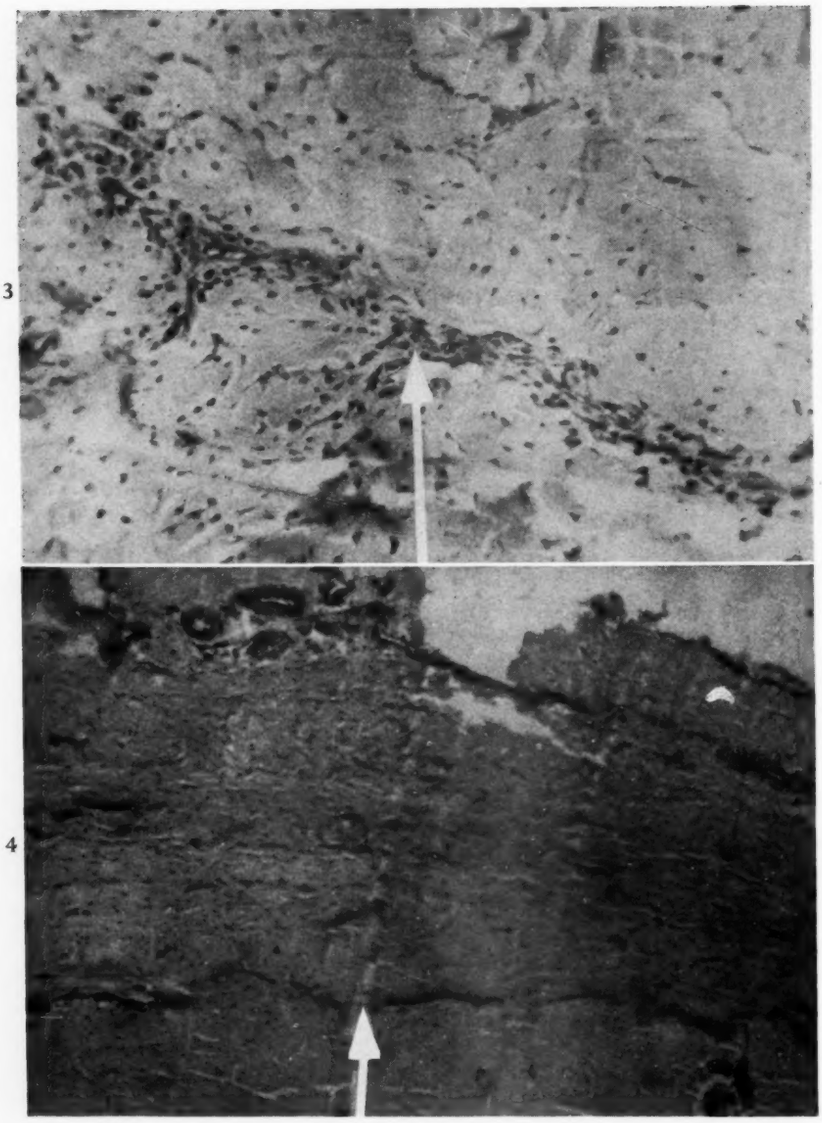


Fig. 3. Fibrous tissue now present. Lymphocytic infiltration minimal. One month after injection. Arrow points to few fibroplasts.

Fig. 4. Fibrosis now present, lymphocytes absent and sheath thickened and fibrosed nine months after initial injection. Arrow points to junction of tendon and its sheath. Nine months after initial injection.

PROLIFERATING SOLUTION

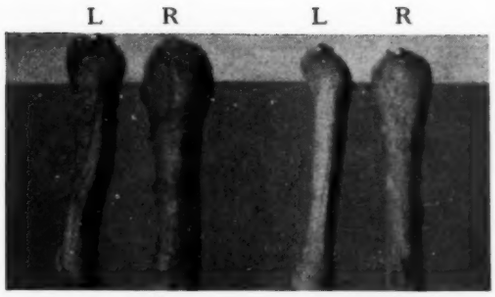
The proliferating solution employed was sylnasol® which has been used clinically by one of the authors in joint stabilization since 1939. Sylnasol,* a mild sodium salt of fatty acid, was chosen because it has been found by other investigators (Rice; Maniol; Harris, White) to have the following desirable features: (1) causes a minimum of early exudate, (2) a minimum of discomfort, (3) stimulates a maximum of permanent white fibrous connective tissue, (4) no systemic reaction, and (5) no sloughing or destruction of tissue.

* Manufactured by G. D. Searle and Company.

METHOD OF INVESTIGATION

The injections of proliferating solution were distributed throughout the tendon from its origin in the muscle to its insertion into the bone.

The second injection was given six weeks after the first, and the third or last injection



9 months 12 months

FIG. 5. Photographs of rabbit tendons nine and twelve months after three injections of proliferating solution were given into the right tendons while the left were used as controls. The right tendon in each case reveals a 40 per cent increase in diameter. The tendons with bone attached at the upper end reveal a 30 per cent increase in diameter as compared with the controls. A 40 per cent increase in diameter is equivalent to approximately 100 per cent increase in strength.

was given five months after the first, so that animals in all cases under two months' duration received only one injection, those under five months received two injections and the longer cases received a total of three injections.

The tissue was stained with hematoxylin and eosin. All the photomicrographs were magnified 200 times. The ones selected for this article show the various stages from immediate acute inflammation through generation of new fibrous tissue to permanent white fibrous tissue.

RESULTS OF EXPERIMENTS

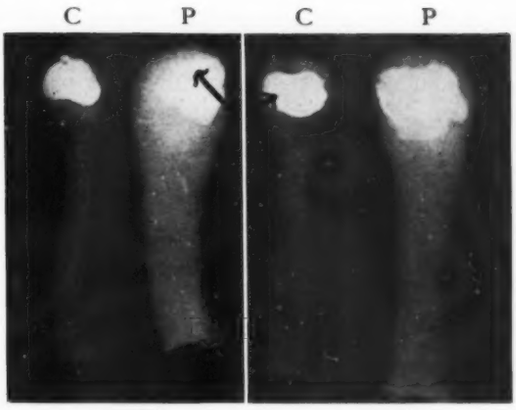
There was no necrosis in any of the specimens and no destruction of any nerves, blood vessels or tendonous bands.

As shown in Figure 1 the early inflammatory process took place surrounding the nerves and blood vessels with lymphocytic infiltration throughout the area between the tendon and its sheath. The inflammation also extended within the junction of the two tendons.

In Figure 2, which was taken two weeks after the original injection, the acute inflammatory process has begun to subside. The lymphocytes are less evident, new capillaries have formed and fibroplastic organization has begun.

Figure 3 show tissue one month after the initial injection. Fibrous tissue is present and lymphocytic infiltration has diminished although some is still present which indicates the production of new white fibrous tissue is still being promoted.

Figure 4 is the section of a nine-month



1 month 3 months

FIG. 6. Arrows denote bone; C indicates control and P, proliferated.

tendon that was injected at six weeks and again at five months after the initial injection. Permanent white fibrous tissue has formed. There is no inflammatory process present and the sheath has been thickened and joined to the tendon.

QUANTITATIVE RESULTS

Figure 5 shows the right and left tendons of two rabbits after nine and twelve months of proliferating treatment. In both cases the left tendon was not injected and was used as a control. The right tendon in each case received three injections. The second injection was given six weeks after the first, and the third injection was made five months after the initial one.

In the nine-month case the tendon sheath remains on both the right and left, and in the twelve-month case the tendon sheath has been removed revealing the normal smooth surface of the control tendon on the left and the rough surface of the right tendon from which the adherent sheath has been forcibly removed.

At the end of nine and twelve months the injected right tendons in each case reveal an increase of 40 per cent in diameter as compared with the left controls, while the end of the bone with the attached tendon discloses a 30 per cent increase in diameter.

Although no mechanical method of computing the relative tensile strength of tendons was available, it is estimated that an increase in diameter of 40 per cent would approximately double the strength of the tendon.

Figure 6 shows the proximal end of the tibial tarsal bone of the rabbit with the attached

gastrocnemius and superficial flexor tendons. The films were made at one and three months after a single injection of proliferant solution had been distributed throughout the fibrous tissue. It reveals soft tissue increase at one month is pronounced due to the presence of inflammatory reaction, while at three months the increase is due to the production of permanent fibrous tissue. It also reveals a marked increase of bone at one month as compared with the control and a further increase of bone at three months.

The increase of bone is significant because it results in a strong fibro-osseous union where sprains, tears and relaxation of the ligament chiefly take place and where the sensory nerves are abundant. It accounts for the excellent clinical results that have been obtained in the past sixteen years in restoring joint stability.

No method has been found to demonstrate whether an actual shortening of a ligament takes place during proliferation or whether the clinical results are due to an increase in strength of the ligament which prevents it from stretching when under strain.

Pain in relaxed ligaments while undergoing strain is due to tension on the nerves within the ligament which do extend as the ligament stretches. The injection of an anesthetic into the relaxed ligament eliminates the pain temporarily, and proliferation of new fibrous tissue eliminates the disability permanently.

COMMENTS

The fibrous bands of joint ligaments crisscross in such a manner that movements of the joint are permitted in different directions, but each movement is restrained within certain limits. When the ligamentous bands become relaxed so that abnormal movement causes discomfort, the injection of a proliferant within the ligament will induce the formation of new fibrous tissue in the spaces between the crisscrossed bands and firmly attached to the bands. The new tissue becomes permanent in a few weeks and inhibits the excessive movements. The joint is thus stabilized, discomfort eliminated and normal activity resumed.

Following injection of the proliferant in any tissue the inflammatory reaction and newly developed fibrous tissue surround the existing nerves and blood vessels, and new capillaries develop as shown in Figure 2 (previously reported by Harris, White and Biskind). This accounts for the vigorous growth and vitality of the permanent new fibrous tissue.

These experiments were carried out in the same manner as the clinical treatments. Local anesthesia was injected ahead of the needle as it was inserted, and the proliferating solution was distributed by injection while the needle was being withdrawn.

Of particular significance is the 40 per cent increase in diameter of the tendon and 30 per cent increase in diameter of the tendon at its bony attachment following three injections of the proliferant as revealed in the photographs of the nine- and twelve-month experiments. It demonstrates the strengthening of the entire tendon including the bony attachment by the production of permanent white fibrous tissue. It is estimated that an increase of 40 per cent in diameter of the tendon would approximately double its strength. It is the opinion of the author that when damage to a ligament occurs it most often takes place at its bony attachment where it is more vulnerable.

Leriche and Gardner have shown that ligaments near their bony attachments are richly supplied by sensory nerves. Pain is present when relaxed ligaments are under normal tension because the fibrous strands of the ligament extend, and the nerves in the ligament are placed under abnormal tension because they do not lengthen.

Some physicians have been getting relief from pain in acute cases of ligament disability by the injection of a local anesthetic. To accomplish enduring stabilization of the joint, and freedom from discomfort and to avoid recurrences when the ligaments have become relaxed, the production of an increased amount of permanent white fibrous tissue is necessary.

Although the original report of ligament proliferation by one of the authors dealt with fourteen years' work on the sacroiliac joint, articles will soon appear describing the technic of stabilizing all the joints of the spine and pelvis.

Arrangements are being made for a source of supply of three-dimensional illustrations of the lumbar spine and pelvis in which the ligaments have been reconstructed on one side with the tender points of ligament relaxation designated. On the other side needles have been placed as in the position of full insertion during treatment. This has been deemed

necessary because for many years the congested medical college curriculum has precluded the medical student from dissecting and otherwise acquiring a familiarity with the ligaments of the spine and pelvis. Therefore, three-dimensional illustrations will facilitate both diagnosis and treatment.

Investigation is in progress on combining the proliferating solution with a long-acting anesthetic which is desirable for the treatment of many ligaments.

During the past fifteen years more than 3,000 injections of a local anesthetic and the proliferant have been given into the dorsal ligaments of the lumbar spine and pelvis. The pressure of the solution has immediately reproduced the local pain confirming the diagnosis. Frequently also reproduced was the referred pain into the groin, buttock and lower extremities to within 2 inches of the ankle but never into the foot. A chart of the referred pain areas will soon be published and will be found useful in the diagnosis of low back disabilities.

SUMMARY AND CONCLUSIONS

1. The injection of a proliferating solution within the fibrous bands of rabbit tendons promotes the production of new fibrous tissue which becomes permanent. The increase in diameter of the tendon was 40 per cent, and the increase in diameter of the bony attachment of the tendon was 30 per cent at the duration of nine and twelve months, although only three injections had been made and none after the first five months. This amount of increase would approximately double the strength of the tendon.

2. New fibrous tissue which develops between and is attached to the crisscrossed bands of the ligaments will curtail the abnormal joint movement.

3. The increased fibrous tissue accounts for the clinical results which were reported by one of the authors in the treatment of 253 cases of sacroiliac relaxation over a period of fourteen years with 82 per cent of the patients permanently relieved of their symptoms.

4. Joint stabilization by proliferation has been successfully employed in the treatment of various joints including all of the vertebral and pelvic joints.

5. No unfavorable incident occurred in either the clinical or experimental cases in which approximately 3,000 injections have been given.

6. Pain in relaxed ligaments is due to tension on the nerves which do not lengthen as do the fibrous bands of the ligament.

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Management of Bronchiectasis in the Older Patient

HELEN S. PITTMAN, M.D., *Boston, Massachusetts*

From the Pulmonary Clinic, Massachusetts General Hospital, Boston, Mass.

BRONCHIECTASIS in older persons is essentially a medical disease. It is a condition which we can rarely expect to cure; rather, the place of the physician is to keep the patient in as good condition and as nearly symptom-free as possible.[1] In most of these patients we are dealing not with bronchiectasis alone but also with variable degrees of pulmonary fibrosis and emphysema. The disease is usually bilateral and quite diffuse, although major areas of involvement may be expected to be found in the lower portions of the lungs. Less commonly the disease is due to a localized bronchial obstructive process. This may be endobronchial tuberculosis, bronchial adenoma, foreign body or very rarely bronchogenic carcinoma. In these situations the bronchiectasis will usually be limited to the segment or lobe distal to the obstruction.

Basic problems of management depend upon the symptoms and the underlying disease.

DIAGNOSTIC PROCEDURES

These should be individualized and not routine, and include the following:

1. *Examination of Sputum.* Routine cultures are worthless as they will reveal a mixed flora.

Search for tubercle bacilli should be carried out carefully when the x-ray suggests past pulmonary tuberculosis or when localized disease could be due to bronchial tuberculosis.

Cytologic examination by Papanicolaou technic is indicated when there is repeated blood streaking. It is, however, often unsatisfactory due to large amounts of pus in the sputum. In bronchial adenoma cytology is not diagnostic.

2. *X-rays.* Ordinary technic postero-anterior and lateral films with the addition of postero-anterior grid film will usually give the necessary information.

Bronchograms should not be made routinely simply to confirm the radiologist's suspicion of bronchiectasis. If, however, surgery is being contemplated, a carefully executed bronchogram is important in outlining the areas of normal bronchial pattern as well as the diseased portions.

3. *Diagnostic Bronchoscopy.* This should be carried out whenever there is a question of bronchial obstruction, inflammatory or neoplastic, and when there is bleeding. The latter is particularly important in the presence of bilateral disease. In such patients bronchoscopy will be of the greatest help in determining which side the blood is coming from if it is carried out while the patient is bleeding.

THERAPEUTIC PROCEDURES

These should be planned in relation to symptoms.

1. *Cough and Sputum.* Cough which helps to raise secretions is useful. A dry, irritating, unproductive cough is useless and measures should be taken to suppress it. In general, one should encourage the raising of secretions during the day. Simple expectorants, such as potassium iodide, syrup of hydriodic acid or terpin hydrate, are usually effective aids to this. When sputum is copious it is important to try to have it raised in advance of each meal so that it will not interfere with eating or cause vomiting from cough. In younger people this is

best accomplished by postural drainage. Older people, however, may be unable to tolerate a head down position because of dizziness. It may be tried cautiously for a very few minutes and, if the patient tolerates the position, it will prove useful. It should be remembered that the function of postural drainage is to start the upward flow of secretions to a point where the cough reflex will be effective in completing the raising of sputum. In this connection it is worth while to mention smoking. Most doctors urge patients with cough to omit tobacco. Certainly excess smoking contributes to coughing. However, it is well to remember that inhaled smoke is often an effective stimulus to cough which raises sputum. A dry cough, particularly one which interferes with sleep, should be suppressed by codeine at night and before meals. A vaporizer or steam kettle at the bedside is useful in controlling irritative cough at night. A DeVilbiss No. 149 runs a full eight hours without attention.

2. *Intercurrent Respiratory Infections.* These people are never free of infection in the respiratory tract. The basic lesion of bronchiectasis is twofold: chronic infection of the tissues of the bronchial wall as well as changes in size and shape of the bronchi. These underlying changes make patients particularly vulnerable to colds, and their colds always are "chest colds" with acute bronchitis at least and frequently areas of pneumonitis. Because of this bacterial reservoir they should receive penicillin, 300,000 to 600,000 U. by intramuscular injection daily, for five to ten days at the start of a cold.

We have found that the number and severity of acute infections can be helped to a worth while degree by the daily use of prophylactic chemotherapy during the season of colds. We start many patients on a sulfonamide (one 0.5 gm., [7.5 gr.] tablet of either sulfamerazine or gantrisin® twice a day) about September 1st and keep them on this daily ration until warm weather is established the following spring. The patients on this regimen are required to return in one week, then two weeks and monthly thereafter during the period of administration in order to have white counts and urine examinations. Any patient who is put on daily chemotherapy should be told what drug he is taking. He should not be given therapeutic doses of sulfonamide for any infection because there is a possibility that it might suppress bone marrow activity and result in dangerous leukopenia.

Instead of a sulfonamide patients may be put on prophylactic antibiotics, penicillin, aureomycin, achromycin,® etc. These preparations are more expensive, they may in some instances induce gastrointestinal upsets and, most important, they may lay the groundwork for serious and dangerous monilial infections.

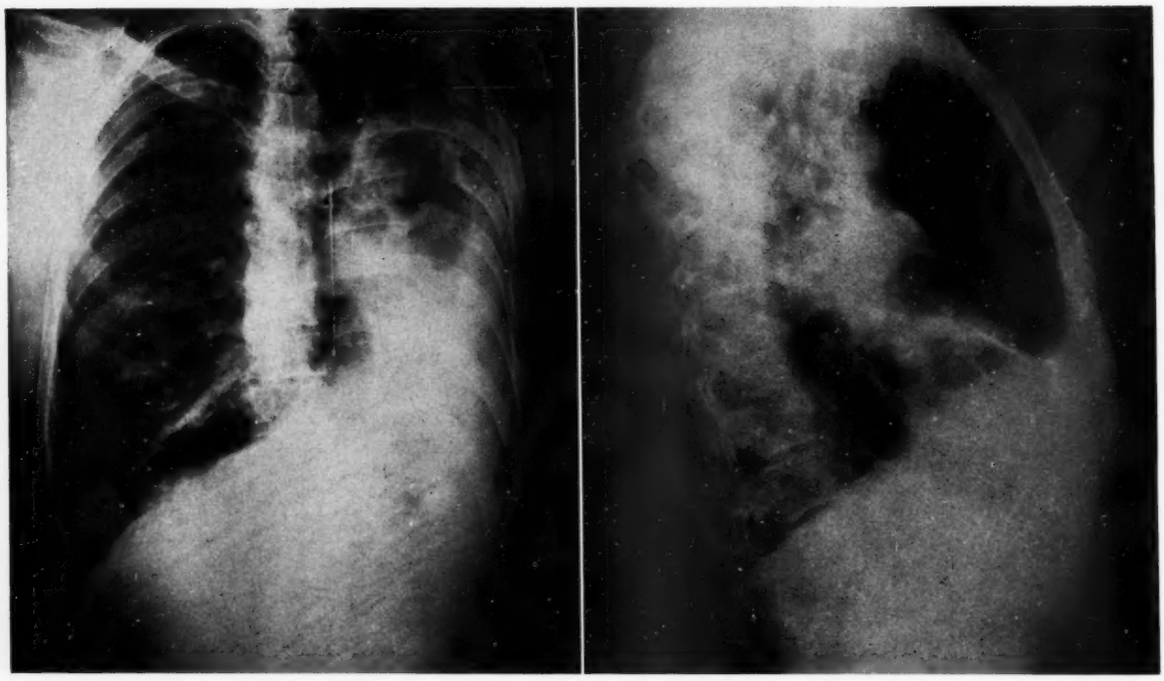
3. *Dyspnea.* This is a troublesome symptom in many patients. It is usually a manifestation of the associated pulmonary fibrosis or emphysema. Spain[2] has produced impressive evidence to suggest that the primary lesion in emphysema is in the terminal bronchiole. He has demonstrated thickening, narrowing, stiffening and inflammation of the bronchiolar walls. According to his ideas this chronic infection, to which recurrent acute infections are added, sets up bronchospasm which causes secondary distention of the alveoli. He notes that emphysema usually occurs in patients with long-standing cough.

Bronchodilator drugs should be used in patients with bronchiectasis who have dyspnea. Aminophyllin by mouth may be tried and is sometimes beneficial. The most useful drugs are vaponefrin, isuprel® and neo-synephrine.® These are administered by nebulizer. (Vaponefrin or DeVilbiss No. 40 are good.) It is important that the patient be taught how to use the nebulizer; it should be inserted in the mouth just beyond the teeth and the spray inhaled slowly.

Oxygen must be used with great caution in the patient with chronic pulmonary disease who is dyspneic and may be cyanotic at rest.[3] These patients probably have both carbon dioxide retention and anoxia. If oxygen is given in an attempt to relieve dyspnea and cyanosis, the increase in arterial saturation which will occur may reduce the anoxic stimulus to breathing. The consequent hypoventilation allows more carbon dioxide retention, and carbon dioxide narcosis may appear. Thus the cyanotic patient who is given continuous oxygen during an ambulance trip to the hospital may arrive in coma.

If oxygen is to be given to these patients, it must be used sparingly and intermittently for short periods several times a day.

4. *Bleeding.* This may constitute an indication for surgery. If hemorrhage is severe, it may be justifiable to make a direct approach on the



1A 1B

FIG. 1. Case 1. A and B, postero-anterior and lateral x-rays taken three years before left pneumonectomy for uncontrolled hemoptysis. Note that although the left lung is the site of major disease, there are also extensive changes in the right lung with emphysema at apex and base and probable collapse of the middle lobe.

source of bleeding even in a poor risk patient. We have recently had such a situation.

PNEUMONECTOMY FOR MASSIVE HEMORRHAGE

CASE 1. (M. G. H. unit No. 102604.) A fifty year old crane operator came to the Emergency Ward of the Massachusetts General Hospital on June 24, 1954, because of hemoptysis for four days.

This man has had a chronic cough since he had influenza in 1918 at the age of fourteen. He was first seen at the hospital in 1926 with hemoptysis and a consequent suspicion of tuberculosis. A diagnosis of bilateral bronchiectasis was made at that time. He had foul breath, cough, sputum and, on one past occasion, had had hemoptysis of one pint. He had lost 35 pounds. Intensive search of sputum failed to demonstrate tubercle bacilli, and the tuberculin skin test was negative. Since then he has been seen irregularly during the past twenty-eight years.

In 1938 a lipiodol examination demonstrated bronchiectasis in left lower, right lower and right middle lobes.

In 1938 he was presented to Dr. Edward D. Churchill for question of operation. Dr. Churchill believed that because of the bilateral involvement surgery was not advisable and advised a series of therapeutic bronchoscopies. Prognosis was considered very doubtful. Bronchoscopy was performed repeatedly with aspiration of large amounts of thick, tenacious, foul secretion from both main bronchi. (This patient is one of those included in the follow-up study made in 1939 by Perry and King.[4])

In 1946 we had begun to use chemotherapy and he was started on sulfadiazine, 0.5 gm. twice a day, together with aerosol penicillin. X-rays at that time were essentially the same as those which were taken on April 23, 1951 (Fig. 1), and interpreted as follows: "There are extensive changes throughout the left lung which is considerably reduced in size. The upper lobe shows numerous areas of rarefaction probably representing bronchiectatic cavities. The area usually occupied by the lower lobe is nearly homogeneously dense. Superimposed on the left lung area is the herniation of the right lung into the left chest. The right middle lobe is probably collapsed. The right lung shows rather diffuse increase in the markings with areas of emphysema in the costophrenic angle and at the apex. In comparison with

previous reports it is rather questionable as to whether there is any real change."

From 1951 to 1954 he continued work and did not report to the clinic. The present hemoptysis was the most severe he had ever experienced, and it continued after admission with falling hemoglobin despite blood transfusions. It was believed that there was now no alternative to operation, and on the fifth hospital day left extrapleural pneumonectomy was performed under endobronchial gas-oxygen-ether anesthesia. The patient received 3,500 cc. of blood during operation. He did well and was discharged in good condition on the twelfth postoperative day.

The pathologic report showed chronic bronchiectasis, chronic lung abscesses with massive hemorrhage into one, and severe chronic pneumonitis with fibrosis.

The patient "felt fine" for four days at home and then an acute sore throat developed. He was readmitted to the hospital on July 14th with a temperature of 101°F., orthopneic and requiring oxygen. X-ray showed areas of density throughout the remaining lung. Throat culture grew beta-hemolytic Streptococcus and Staphylococcus aureus. He was critically ill for about three weeks and then began to improve. At the present writing (August 19, 1954) he is making a good recovery, is afebrile, ambulatory, and current x-rays show that the process has cleared markedly.

The patient was seen in the clinic on September 15, 1954, three weeks after discharge. He had no subjective shortness of breath and no cough. There were a few coarse rales in the right lung. He had been on penicillin and gantrisin in therapeutic doses. These have been omitted but he will take gantrisin, 0.5 gm. twice a day, through the winter.

The patient received penicillin and streptomycin for eight days after operation, the last dose being given four days before discharge. In view of the known bronchiectasis in the remaining lung it would undoubtedly have been wiser to continue antibiotics beyond the usual period. The postdischarge infection, however, started in his throat and was probably exogenous. Electrophoretic pattern showed that he has adequate amounts of gamma globulin in his serum.

This case is reported to show how, after years of medical management in a patient rejected for surgery because of the extent of his disease, operation may be forced as a life-saving measure.

MANAGEMENT OF NON-PULMONARY SURGICAL PROCEDURES IN PATIENTS WITH BRONCHIECTASIS

Necessary surgery may be carried out under local or general anesthesia. The lungs should be made as secretion-free as possible by the previously mentioned measures to control cough and sputum. Antibiotics should be given. A preoperative aspiration bronchoscopy is the most effective way of clearing wet lungs in a patient who does not raise efficiently. For a long surgical procedure under general anesthesia we like intratracheal ether with bronchoscopic aspiration at the conclusion of the procedure.

CASE II. (M. G. H. unit No. 848483.) A seventy-one year old woman was admitted on May 26, 1954, to the private service of the Massachusetts Eye and Ear Infirmary for cataract extraction and was referred for preoperative evaluation because of her bronchiectasis. The patient could not recall the onset of her cough; her daughter, a physician, stated that she had raised foul sputum as long as anyone in the family could remember. For many years she had been known to be hypertensive and for at least twenty years had had exertional dyspnea, six-pillow orthopnea and frequent ankle edema. In addition she had been found three years before to have diabetes, and it had never been well controlled.

On examination the patient was very short, about 4 feet 10 inches, and weighed 166 pounds. The breath was foul, she had a loose, wet cough and spat ineffectively. The sputum was mucopurulent, foul and contained considerable amounts of red blood. The chest was emphysematous with marked dullness and many coarse rales at the right base. The heart was rapid with occasional premature beats, slightly enlarged and the sounds were of poor quality. Blood pressure was 190/100.

Electrocardiogram was interpreted as showing cardiac irritability and changes consistent with hypertensive heart disease. Fasting blood sugar on admission was 318 mg. per cent, non-protein nitrogen 70 mg. per cent. Papanicolaou examination of the sputum was unsatisfactory because of the very large amount of pus in it.

Chest x-ray taken May 27th was reported as follows: "The heart is moderately enlarged

and prominent in the region of the left ventricle. The aorta is tortuous. There is irregular mottled density within the basal portion of the right lower lobe apparently also extending into the middle lobe. There is some loss in volume of these portions of the lung. Interspersed with the zones of density are a number of irregular cavities and evidence of cylindrically dilated bronchi. Above the gross area of increased density and adjoining the right hilus is a nodular 2 cm. density, the precise nature of which is not certain. It may represent a large vessel or a parenchymal nodule. The vascular markings in the left lung are somewhat prominent. There is evidence of pleural reaction at the right base which may be due to either thickened pleura or a small amount of fluid."

"Impression: Cardiac enlargement with left ventricular predominence. Atherosclerotic changes of the aorta. Mottled density within partially atelectatic right middle and lower lobes probably representing pneumonitis and bronchiectasis. It is not possible to exclude an intrabronchial obstructing lesion as responsible for these changes, either a carcinoma or adenoma."

It was considered that the patient had severe bronchiectasis with retention of secretions and limited pulmonary and cardiac reserves. Aspiration bronchoscopy was advised, but because of family opposition it was not performed. She was put on a regimen of antibiotics and expectorants; and since she was said to be sensitive to penicillin and aureomycin, 250 mg. of terramycin® was used every six hours, with 4 cc. of terpin hydrate every four hours. Her diabetes was brought under better control with adequate insulin, salt was restricted and she was digitalized.

On the ninth hospital day, after preparation with nembutal® and demerol®, surgery was performed under local anesthesia. The next day examination of the chest showed flatness with absent breath sounds at the right base; this was interpreted as indicating bronchial occlusion from retained secretions following her heavy sedation with suppression of cough on the day of operation. The maximum postoperative temperature was 99.2°F., and as the patient became more active her signs of atelectasis disappeared.

She was discharged on the fourth postoperative day with the eye in excellent condition. Her pulmonary status was unchanged, the CO_2 content was 32 mEq./L. (normal 22.5 to 29.2) and her diabetes had been regulated. Fasting blood sugar on the day of discharge was 154 mg. per cent, non-protein nitrogen 38 mg. per cent.

This case is reported as an example of medical management of bronchiectasis whereby the satisfactory performance of elective eye surgery in an elderly, poor risk patient was made possible.

SUMMARY

1. Bronchiectasis in the older patient is usually secondary to other disease in the chest and should be managed medically.

2. Treatment is aimed at conserving function and is directed at control of symptoms.

3. Measures aimed at reducing intercurrent infections are outlined.

4. Caution is urged in the use of oxygen.

5. Surgical interference may be justified in the presence of severe hemorrhage. Such a case is reported.

ADDENDUM

The patient in Case 1 has been followed in the clinic regularly since his discharge from the hospital and was last seen on March 3, 1955. On December 1, 1954, he resumed his occupation full time and has worked steadily. He continues to have a few rales at his right base, the site of known bronchiectasis, and he coughs to raise a trace of mucus each morning. Sulfamerazine, 0.5 gm. twice a day, has been continued without interruption. X-ray of his right lung shows no change from the preoperative examination. He has regained all but 5 pounds of the weight lost during pneumonia.

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Bronchiectasis

FRANCIS M. WOODS, M.D., *Brookline, Massachusetts*

From the Overholt Thoracic Clinic, Boston, Massachusetts.

BRONCHIECTASIS means dilatation of bronchi; this is a pathologic situation usually associated with chronic infection and involving the secondary and smaller bronchi. It is the result of a variety of diseases. Although described by Laennec in 1808, its importance came to light in the last three decades, with the diagnostic use of iodized oil. No sharp line can be drawn between a normal and a dilated bronchus nor between the uninflamed and the chronically infected bronchus. Nor can any clear distinction be made between a dilated suppurative bronchus and a lung abscess with communicating bronchus. The changes may involve the bronchi only or they may spread out into the adjacent parenchyma. The bronchial walls may be diffusely dilated into tubular types or irregularly dilated into saccular types. The muscle, elastic and cartilage components may be partially or completely replaced by fibrosis and inflammation. If the fibrosis spreads out into the parenchyma, the involved segment may be more or less contracted and consolidated, and the term atelectasis is commonly though incorrectly applied to such fibrotic contracted segments. The dependent segments are the only ones involved in most instances. Thus the basal segments of the lower lobes, middle lobe and the lingula are the usual sites. Any combination of these segments may be involved. The anterior segment of the upper lobe and the superior segment of the lower lobe come next in frequency.

ETIOLOGY

Ordinarily the cause of a given case of bronchiectasis cannot be ascertained. The history is usually much more prolonged than that of tumors or foreign bodies in the bronchi, but it is known that bronchiectasis does develop beyond such obstructions. It seems logical, therefore, that some partial obstruction preceded the development of bronchiectasis. Lymph glands are often seen surrounding and partially compressing the base of dilated groups of bronchi, but whether the lymph glands resulted from a preceding infection or caused infection in the obstructed bronchi is impossible to say. A history of protracted pneumonia, tuberculosis or pertussis is not unusual. Two factors, obstruction and infection, seem to be necessary. The usual distribution in the dependent bronchi suggests that gravity acts as a significant additional obstructive factor.

SYMPTOMATOLOGY

There is no consistent correlation between the extent of the symptoms and the extent of the disease. Characteristically, there is chronic cough productive of small or large amounts of green or yellow globules of sputum. In severe cases it may be fetid. Superimposed episodes of frank pneumonia or, at least, bronchitis with fever, malaise and increased cough and expectoration are common. Hemoptysis is far commoner than previously supposed. In the absence of roentgen evidence of parenchymal disease in the lung (and assuming that heart disease and hypertension and blood dyscrasias are excluded) bronchiectasis is the most likely cause. Strangely, hemoptysis due to bronchiectasis frequently occurs unassociated with cough or sputum and it is then referred to as "dry bronchiectasis." Dyspnea suggests extensive involvement or related fibrosis and emphysema. Clubbing of the fingers, weight loss, weakness and inanition are usually seen in advanced cases. It is important to realize that minimal demonstrable bronchiectatic changes may be associated with considerable symptoms and conversely extensive changes may occur with few, if any, symptoms.

DIAGNOSIS

Positive diagnosis depends on bronchography. However, physical examination may reveal rales in the distribution of dilated bronchi. These may be inconstant, appearing only with clinical exacerbation. Dullness and decreased breath sounds may be noted in contracted

areas. Plain chest x-ray reveals no abnormality if the inflammation does not extend beyond the bronchi, but in more advanced cases there are increased lung markings or densities where fibrosis and inflammation extends out into the parenchyma.

In order to visualize bronchi they must be outlined with contrast media just before x-rays are taken. This procedure is called bronchography. Our technic consists of anesthetizing the pharynx with a spray of 10 per cent cocaine. Premedication should include a barbiturate. Five to 10 cc. of 2½ per cent cocaine is dripped into the trachea leaning the patient to the left for part of the injection to assure anesthetizing the left bronchus. A single rubber catheter (No. 18) is passed between the vocal cords and the patient lies on the tilting fluoroscopic table. The tip of the catheter is placed beyond the carina on the side to be studied first. Forty per cent lipiodol is injected through the tube slowly. The patient is rotated and tilted and the tube is moved in any convenient sequence to allow outlining of all the segments on the side to be studied. Immediately the patient stands and a postero-anterior and lateral x-ray is taken. In adults 6 to 10 cc. of lipiodol is sufficient to outline one side; more may "flood" the alveoli and result in obscuring the bronchi. Rapid coordinated steps result in the best bronchograms, for the lipiodol may diffuse rapidly into alveoli. After the first side is done, the tube is changed to the opposite side which is outlined in the same way. Now, postero-anterior and both obliques are taken—the latter at 30 degrees. The catheter is withdrawn and the patient is encouraged to cough up the oil and is placed in the Trendelenburg position to promote drainage of the lipiodol. All films are full-sized, 6 foot plates, since spot films waste time and do not give comparison of normal and dilated bronchi. Most children under eight years cannot cooperate sufficiently, therefore, they are given deep ether anesthesia. An endotracheal tube is inserted, and a polyethylene tube passed through the endotracheal tube is used for the introduction of lipiodol. The procedure is otherwise as in adults except that the x-ray films are taken in a recumbent position. At the close of the procedure the bronchial system is easily cleared by suction.

In the near future water soluble media may well replace lipiodol. Although not fully perfected, they have the advantage of being rapidly eliminated whereas lipiodol may adhere to the bronchi or alveoli for many weeks. Pulmonary resections within three to four weeks of lipiodol bronchography are more likely to be complicated.

At the time of operation additional evidence can be obtained concerning abnormal segments which may not have been recognized in the bronchograms. Bronchiectatic segments may feel crepitant and inflate or deflate perfectly normally, but usually there is some induration and/or nodulation in an abnormal segment and inflation and deflation is retarded. Marked delay in deflation is called air-trapping.

TREATMENT

Bronchiectasis as a general rule does not progress from segment to segment. Treatment is, therefore, based on extent of symptoms. The extent of symptoms does not necessarily correlate with the extent of anatomic change. Thus it may be that minimal bronchographic change may require radical treatment.

Prevention. As always, prevention is the best treatment. Adequate and prompt care of all pulmonary and bronchial infections, particularly pertussis in children, pneumonia and tuberculous infections, should reduce the incidence of bronchiectasis. Foreign bodies in the bronchus should be removed promptly bronchoscopically and, if necessary, surgically.

Non-surgical. Non-surgical methods are recommended for the control of exacerbations of symptoms, for preparation for surgery, and in postoperative recovery. In mild cases they are in themselves adequate. In widespread cases they are used as the best palliation possible. They are mechanical and antibiotic and chemotherapeutic.

Mechanical methods promote better drainage of secretions and improve alveolar ventilation. Cough should not be suppressed by medication, but encouraged since it is the normal method of expulsion of secretions. Medications to decrease the viscosity of secretions such as ammonium chloride, potassium iodide and other expectorants are useful in making cough more effective. Inhalation of detergents such as allevaire, which may be given via a nebulizer, are very valuable. Positioning the patient with the head hanging low over the side of a bed or table is called postural drainage. Often, a patient by taking a few minutes of postural drainage once or twice a day will

evacuate his secretions so well as to eliminate cough for the rest of the day. In severe situations it may be necessary to aspirate secretions directly from the bronchial system either by transtracheal catheter or bronchoscopy.

In the event of oxygen want, oxygen is administered by simple, nasal catheter, mask or tent. The catheter is preferred as effective, simple, and interfering least with patient comfort and nursing care. Intermittent positive pressure oxygen administered through a Burns valve* has proved invaluable in more severe oxygen want. This valve administers oxygen under regulated pressure with inspiration. With expiration the pressure drops down to atmospheric allowing complete exhalation. Since a larger volume of oxygen is forced into the lung, there is a more satisfactory force for the expulsion of secretions. Since exhalation is unimpeded, no carbon dioxide accumulation takes place. Alevaire and antispasmodics may be nebulized through a parallel system with these valves.

Antibiotic and chemotherapeutic agents are used to control acute infections and minimize chronic ones. Broad-spectrum drugs are usually indicated because the flora of the bronchial system is likely to be mixed. Vigorous therapy during acute episodes is indicated and the drug or drugs used should ideally be chosen after sensitivity study. We hesitate to recommend indefinite use of antibiotics and chemotherapeutics because of the danger of superinfection.[1] Prolonged antibiotic and chemotherapeutic treatment in low doses may be justified in certain instances of widespread disease with severe symptoms. The status of this question is not yet clear.

Surgical. Excision of all pathologic segments is the ideal treatment and offers the best probability of permanent relief of symptoms. It is undertaken only after the best preliminary improvement has been obtained by non-surgical methods. If bronchiectasis is completely localized to excisable segments, complete relief can be expected. Compromise with the principle of complete excision is indicated, however, when bronchiectasis involves so many segments that excision of all would lead to significant pulmonary insufficiency. Compromise may also be indicated if the segments not involved by bronchiectasis are poorly functioning because of asthma, emphysema or other abnormality. Obviously, younger people will tolerate greater pulmonary tissue loss than older individuals with lesser cardiac and pulmonary reserve. Less functional loss will be sustained in removing completely destroyed segments than segments which are still functioning to some degree. Pulmonary function studies may help to evaluate borderline cases, but gross observations such as ease of climbing stairs, use of accessory respiratory muscles, the degree of emphysema, and general vitality of the patient are all useful. In less than ideal cases one may compromise by only excising the worst segments or in severe situations by depending only on postural drainage, expectorants and antibiotics.

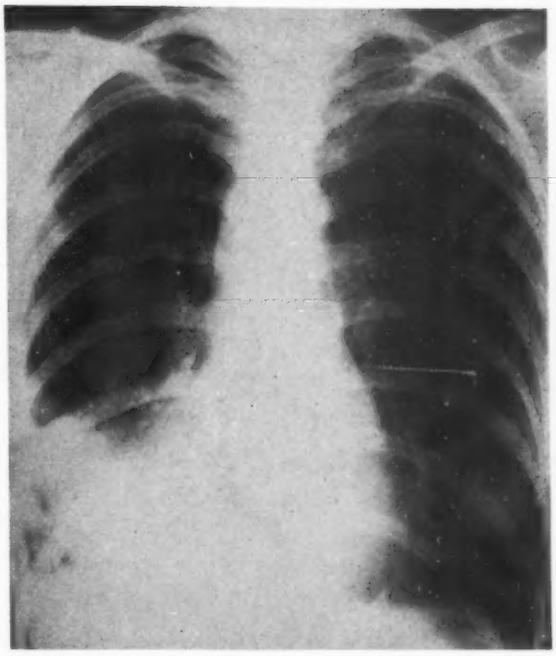
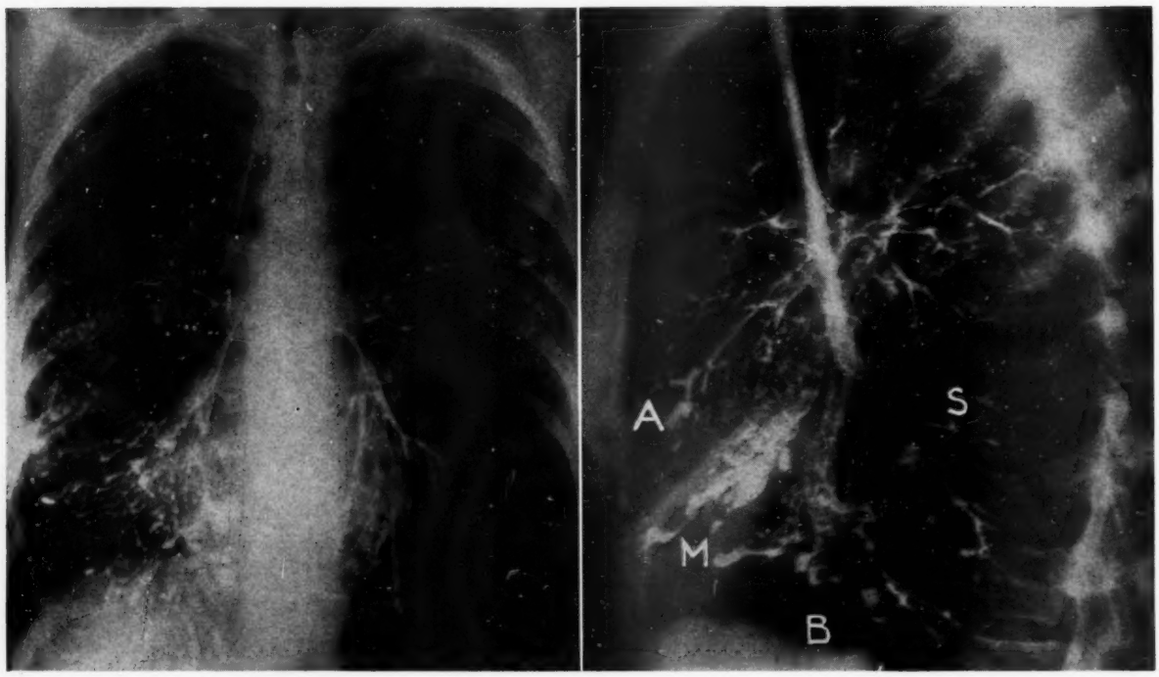


FIG. 1A. Case 1. Plain chest x-ray showing density in the lower lung field caused by bronchiectasis with inflammation extending out into the lung parenchyma.

At operation, the thorax is opened on the side of involvement. If the disease is bilateral, the side of greatest involvement is opened first. The lung is completely freed of adhesions and before excision is started all the evidence is evaluated. First, what areas of bronchiectasis can be determined by the bronchogram? Second, what segments are indurated, nodular or covered by dense adhesions? Third, what areas

* Available through the Mine Safety Appliance Company, Pittsburgh, Pa.; the Emerson Company, Cambridge, Mass.; and V. Roy Bennet and Associates, Los Angeles, California.



1B 1C

FIG. 1B and C. Case 1. B, bronchogram showing dilated saccular bronchiectasis in the lower part of the right ling field. C, lateral view of the bronchogram with the right side only filled. This shows bronchiectasis involving primarily the basal segments of the right lober lobe (*B*) and the middle lobe (*M*). There is minimal involvement of the anterior segment of the upper lobe (*A*), and the superior segment of the lower lobe (*S*) is normal.

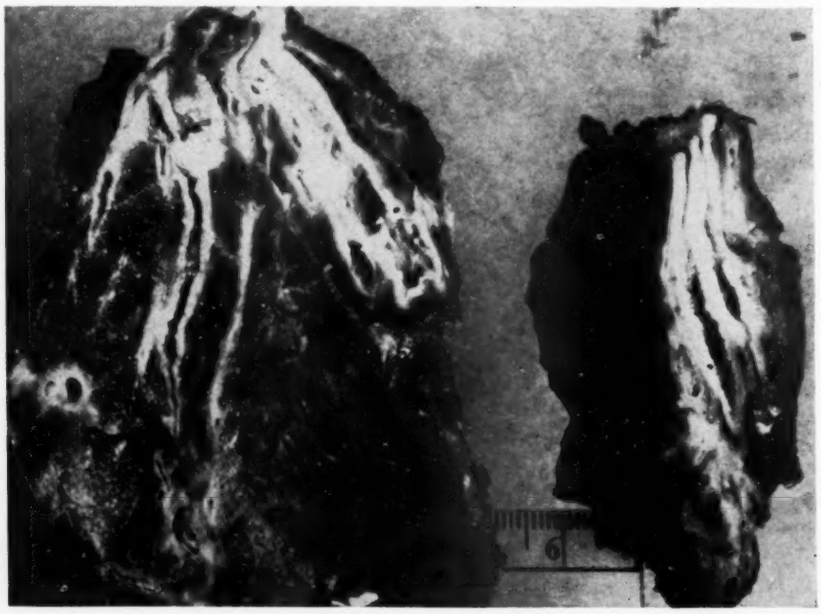


FIG. 1D. Case 1. Resected middle lobe on the right and the basal segments of the lower lobe on the left showing markedly thickened and dilated bronchial tubes with extension of the inflammation into the parenchyma which is fibrotic and contracted.

trap air inflating slower and deflating slower than the normal segments as the anesthetists assist respiratory exchange? Finally, in what areas have rales been heard and pneumonitis occurred?

All diseased segments are removed using individual hilar technics whether total lobes or segments are to be removed. Occasionally, a small diseased subsegment can be simply removed by the less anatomic clamp-and-cut technics. The perfection of technics of segmental resection in the hands of Overholt and his co-workers[2] and others has led to the salvage of many normal functioning segments. Since the basal segments of the lower lobes are commonly involved and the superior segments usually not involved, the latter constitute an important salvage. That such salvaged segments function is suggested by the studies of Overholt, Walker and Etsten[3] and confirmed by clinical observation of such patients.

Segmental resection implies salvage of normal segments and increases the possibilities of bilateral excision. Bilateral simultaneous excision has been done in very good risk patients with well localized problems, but is not an established procedure. Usually, an interval of several months should occur between sides. Actually, many patients experience such relief after the first operation that often the second one can be indefinitely postponed.

CASE REPORTS

CASE I. H. B., a thirty-five year old housewife, had pneumonia four times in eleven years. There was a long-standing history of chronic cough and purulent expectoration with marked increase in the year before operation. Blood-streaked sputum and wheeze had occurred. She had always been malnourished. Examination revealed many rales, front and back, in the lower right chest. Plain chest x-ray (Fig. 1A) showed density in the right lower lung field. Bronchograms in the postero-anterior and lateral view (Fig. 1B and C) showed advanced saccular bronchiectasis in the middle lobe (*M*), and the basal segments of the lower lobe (*B*). The superior segment of the lower lobe (*S*) was normal as was the left side. The anterior segment of the upper lobe (*A*) showed questionable dilatation. At thoracotomy in May, 1951, the basal segments of the lower lobe and the middle lobe were removed. The anterior segment of the upper lobe felt normal and did not trap air; it was not removed. The patient's vitality, weight and strength have all improved. Her respiratory symptoms have virtually disappeared.

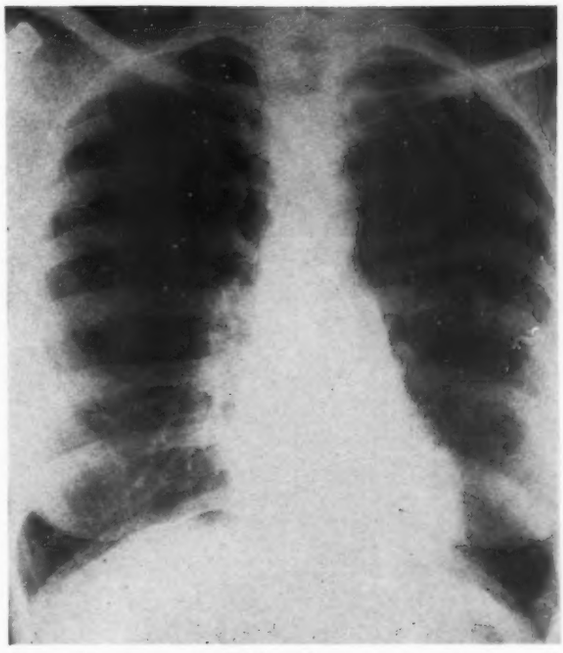
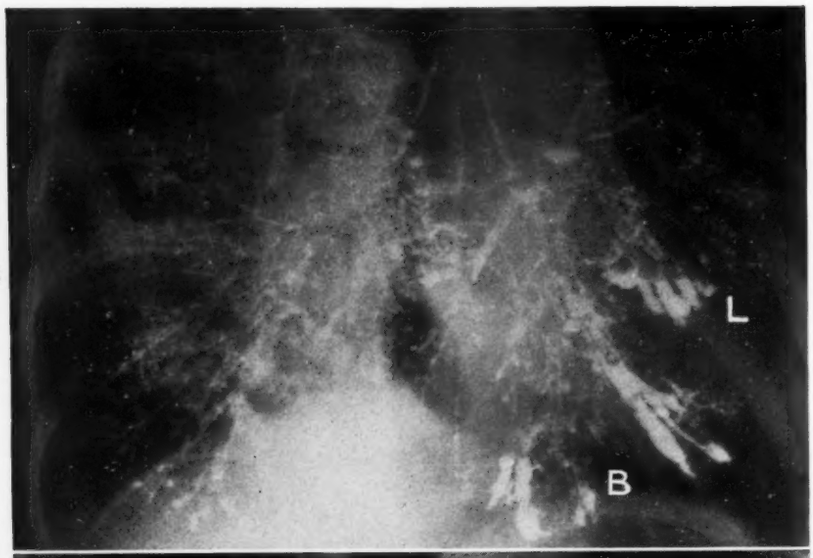


FIG. 2A. Case II. Plain chest x-ray showing advanced bronchiectasis. The plain x-ray of the chest is clear.

Comments. This represents a classic case of well localized bronchiectasis which can be safely eliminated. The danger of future trouble is minimal. Figure 1D shows on the left the basal segments. Note the marked thickening and irregularity of the bronchi which do not narrow as they reach the periphery. The middle lobe on the right shows even more advanced involvement with extension of the fibrosis into the parenchyma. This is the cause of the density in the plain x-ray film.

CASE II. M. L., a twenty-six year old housewife, gave a long-standing history of cough, purulent sputum, minor hemoptyses and many respiratory infections. Examination in 1952 showed rales at both lung bases, front and back. Plain chest x-ray (Fig. 2A) was essentially clear. Bronchograms showed advanced saccular bronchiectasis of the lingula (*L*) and the basal segments of the left lower lobe (*B*), seen best in the right anterior oblique films. (Fig. 2B.) In the left anterior oblique film (Fig. 2C) bronchiectasis of the middle (*M*) and the basal segments of the right lower lobe (*B*) is best seen.

2B



2C

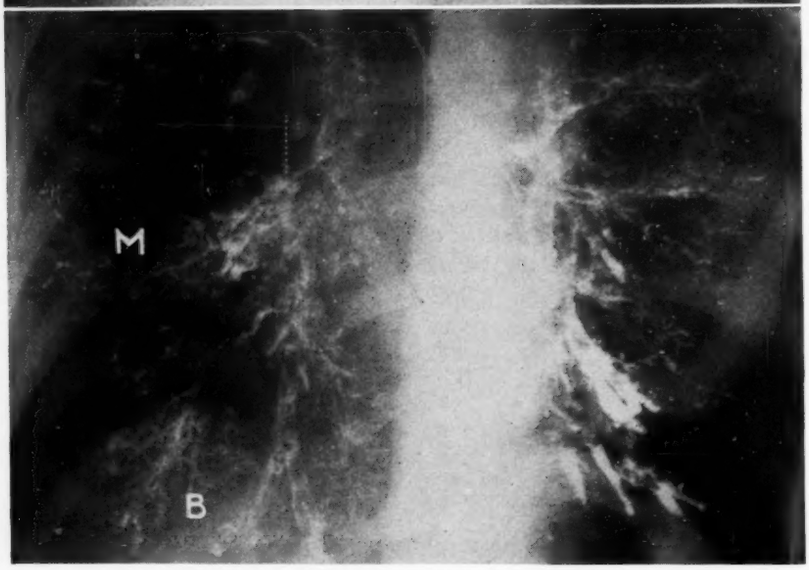


FIG. 2B and C. Case 11. B, oblique views showing advanced bronchiectasis in the lingula (*L*) and in the basal segments of the left lower lobe (*B*). C, left anterior oblique view shows moderately advanced bronchiectasis in the middle lobe (*M*) and the basal segments of the right lower lobe (*B*). Also, this view shows the advanced bronchiectasis already described on the left side. Despite advanced bronchiectasis, it was not necessary to operate upon this patient who had few symptoms.

The patient had never received any therapy whatsoever. She was placed on postural drainage and given an intensive short course of broad-spectrum antibiotics and expectorants. When seen two years later her symptoms remained so minimal that excisional therapy was not contemplated.

Comments. It is unusual for such advanced bronchiectasis to be easily controlled but it is to be emphasized again that extent of bronchographic changes and symptoms do not necessarily correspond. Surgical intervention is recommended for patients with recurring or intractable symptoms providing that bronchiectasis is sufficiently localized.

SUMMARY

Bronchiectasis means dilatation of bronchi and is usually associated with chronic cough,

expectoration and frequent respiratory infections. Permanent relief can be offered those patients whose disease is reasonably localized, by surgical excision. Postural drainage and antibacterial therapy offer temporary relief. Therapy is guided by extent of symptoms and not by extent of bronchographic changes for the two do not necessarily correspond and bronchiectasis does not ordinarily spread from segment to segment.

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Grinnell's studies on forty patients with squamous cell carcinoma of the anus showed a surprisingly greater incidence of spread upward to the perirectal and mesocolic lymph nodes (along the superior mesenteric artery) than a lateral spread to the axillary nodes. Furthermore, these latter were involved later when the disease was more advanced. Previously radiotherapy had been depended upon, principally as a palliative therapy. Now, with earlier diagnoses based on prompt biopsy reports, abdominoperineal resection is preferred as affording the best possibility of a cure. Radiotherapy produced anal strictures and surely could not affect any involved pelvic glands. (*Richard A. Leonardo, M.D.*)

Acute Lung Abscess

WITH EMPHASIS ON MEDICAL ASPECTS

S. Aubrey Gittens, M.D. and John P. Mihaly, M.D., *New York, New York*

From the Medical Service of the Harlem Hospital, New York, New York.

Lung abscess is a localized area of suppuration in lung tissue, with or without cavitation, and accompanied more or less by necrosis. Pulmonary gangrene is a massive fulminating abscess, differing from the latter in degree but not in kind. While time is the most convenient factor in distinguishing acute from chronic abscess, the dividing line is somewhat hazy. Usually, a duration of less than six weeks earmarks the abscess as acute. A subacute abscess is one which has been present seven to twelve weeks, and a duration of greater than twelve weeks is accepted as evidence of chronicity.[1] One must not, however, be blinded by chronology in assessing the stage of disease in any given case. Judgment must be based on the total clinical and radiologic picture and should be very critical since an abscess with chronic features cannot be cured medically.

BACTERIOLOGY

By common concept, lung abscess is a lesion caused by one or several species of pyogenic bacteria. Extraordinary fungous, acid-fast and parasitic infections constitute a special category.

In spite of apparent initial specificity, bacterial abscesses usually become complicated by secondary invasion and a large variety of organisms can often be recovered from the sputum by proper bacteriologic technics. Besides the ubiquitous streptococcal and staphylococcal floras, the most notorious secondary invaders are the normal inhabitants of the oropharyngeal flora, such as pneumococci, Bacillus welchii, fusiform bacilli, oral spirochetes, and other miscellaneous anaerobes. Hemophilus influenzae, Escherichia coli, Bacillus tularensis and even Bacillus mallei (glanders bacillus)[2] have been recovered.

When the infection is caused predominately by anaerobic organisms, putrefactive necrosis occurs and gives rise to the characteristic fetid sputum and breath. A "foul-smelling sputum," however, is not a prerequisite for a diagnosis of acute lung abscess since it will not be encountered in aerobic infections of the lung with abscess formation.

Lung abscess is definitely decreasing in incidence, perhaps because of a more liberal administration of the various antibiotics early in the course of any infectious disease. Thus potential suppuration, in the lungs as elsewhere, is usually aborted. Higher nutritional standards and better oral hygiene are also contributing factors in reducing the number of cases.

SITE

The right lung is more frequently involved than the left, perhaps because of the more vertical course of the right main bronchus. In our studies at the Harlem Hospital the right lung was involved in 70 per cent of the cases.[3] In spite of previously accepted belief, the lower lobes are not the most common sites. Although over 50 per cent of the aspirational abscesses are located in the apices of the lower lobes,[4] we have found acute abscess from all causes to be equally distributed between upper and lower lobes. Some authors, moreover, have found the upper lobes to be far more common sites of abscess formation.[5] These findings may be explained by the greater accessibility of the upper lobe bronchi to aspirated material when the body is in the supine or lateral recumbent position, as in sleep or unconscious states.

PATHOGENESIS

A study of the pathogenesis is not merely academic but will inevitably shed a revealing light on the best possible treatment. A mere suppression of pyogenic activity by antibiotics is self-delusory where underlying causes have not been detected and removed so as to prevent their recurrence. The following modes of

pathogenic evolution are recognized and presented in approximate order of frequency.

Aspirational-Inbalational. Because of the nature of this type of abscess it always communicates with a bronchus, although such communication may be temporarily obliterated by bronchial occlusion. Aspirational abscesses are most generally seen in those conditions in which the cough reflex is depressed or abolished, as in acute alcoholism; debilitated, stuporous and comatose states; neurologic disorders; drug addiction; general anesthesia; and occasionally in deep sleep promoted by soporifics. Although dental and oropharyngeal surgery is particularly prone to complication by acute lung abscess, any type of surgery, far removed from the upper respiratory passages, may be responsible as a result of the accompanying deep anesthesia. Regurgitation of gastric contents is likely to cause very destructive necrosis because of the digestive enzymes and acid content of the gastric juice. Cardiospasm and esophageal overflow are occasional factors.[6,7] Frequently concealed by the patient are such predisposing causes as unconsciousness following an altercation or electroshock therapy.[8] Foreign body inhalation, especially in children, and water aspiration, particularly sea water, may be responsible.

Neoplastic. Secondary infection in pulmonary tumors, particularly carcinomas, is not an infrequent finding. Necrosis and suppuration may involve the primary growth *per se*. More common is suppuration in atelectatic lung distal to the neoplasm. The bronchial obstruction caused by neoplasms interferes with adequate drainage and with other defensive pulmonary dynamics, allowing a nidus of organisms to flourish in the secretions which have collected distal to the obstruction. Other patterns of spillage are possible, as described by Brock.[9] Carcinoma should always be considered as a ranking possibility as a cause of lung abscess in an individual over forty years of age. An intensive and persistent diagnostic search must be made in every case to rule out carcinoma as the primary cause in spite of preliminary negative data.

Postpneumonic. Some types of pneumonia may proceed to abscess formation. Pneumonitis caused by Klebsiella pneumoniae (Friedländer's bacillus) is of a type sufficiently peculiar even in its early stages to make the distinction between this type of boggy pneumonitis and pulmonary suppuration extremely difficult. With inadequate therapy it may fail to resolve and proceed to frank abscess formation. Small abscesses are encountered early in the course of staphylococcic pneumonia. Streptococcal pneumonias are usually suppurative from the onset. Lung abscess secondary to pneumococcic pneumonia is rare except in infancy and senility.

Traumatic. Penetrating wounds of the thorax open an obvious route for pyogenic infection of lung tissue. However, closed injuries due to direct or contra-coup blows may result in secondary infection of intrathoracic hematomas, which act as excellent culture media for organisms which may have been aspirated concomitantly or subsequently, or which may have been circulating in the blood stream. One should not overlook the possibility of abscess merely because an open chest wound has not been demonstrated.

Direct Extension from Adjacent Organs. The most frequent primary foci of such invasion are pyogenic infections of the esophagus, spine and subphrenic abscesses. While empyema is not an infrequent complication of acute lung abscess, it must not be overlooked that the reverse can also occur.

Septic Embolism. Since blood from all parts of the body must be necessarily funneled through the pulmonary circuit, septic emboli arising from any focus in the body will be filtered through the vascular bed of the lungs. Abscesses of embolic origin tend to be multiple,[4] and are often associated with infarct formation. Septic thrombophlebitis of the lower extremities is a very common primary focus.

Miscellaneous. Multiple small abscesses are not infrequent late in the course of chronic bronchiectasis, and may coalesce to form a large acute abscess. Various types of localized pulmonary disease such as atelectasis, fibrosis secondary to granulomatous disease, and chronic non-specific pneumonitis may occasionally flare up in acute abscess formation. Secondary suppuration may arise in bland pulmonary infarcts occurring on a cardiac or some other non-infectious basis.[10] Lung abscess secondary to skin trauma has also been described.[11]

COMPLICATIONS

Complications may be divided into those caused by the rampage of the abscess and those

arising as a result of ineffective healing. In the former category are (1) spillage of infectious material through the bronchial tree giving rise to daughter abscesses in ipsilateral or contralateral lung; (2) rupture of abscess into the interlobar fissure or pleural cavity, causing localized or generalized empyema; (3) rupture both into bronchial tree and pleural space, causing bronchopleural fistula and pyopneumothorax; (4) erosion of blood vessels, especially bronchial arteries, causing hemorrhage;[16] and (5) metastatic implantation of infectious material into other organs, particularly the brain. In the second category are (1) lapse of an acute abscess into chronicity; (2) residual bronchiectasis remaining as a focus for future suppuration; and (3) extensive fibrosis, resulting in the clinical syndrome of pulmonary fibrosis and emphysema and causing an embarrassed pulmonary circulation and ventilatory crippling.

Medical management in most of the foregoing complicating features of lung abscess can rightfully be confined only to optimal preparation for adequate surgery.

TREATMENT

Before the advent of the antibiotics and more precise methods of physiologic evaluation, medical management was largely of a palliative character. In a large number of cases, as high as 40 to 60 per cent in some series, the process lapsed into chronicity, became complicated and ended as a surgical problem.[3] Volatile oil inhalations, vaccines, intravenous alcohol and x-ray therapy were tried, but proved of little value. Arsenicals were given where spirochetes were recovered, and potassium iodide when fungi were demonstrated, but both these classes of organisms were usually only secondary invaders. Recovery was obtained with such measures alone in a substantial number of cases, yet as high as 35 per cent of these patients terminated fatally while apparently recovering. Lung abscess was therefore considered a "surgical disease" from the onset, external drainage being the treatment of choice, since resectional surgery had not yet come of age. The antibiotics, however, brought changing concepts of treatment. It is true that uniformity of results and unanimity of opinion still does not exist; nevertheless the pendulum is definitely swinging to the belief that acute uncomplicated lung abscess is primarily a medical disease.[3]

Successful medical management depends on (1) prompt and adequate antibiotic therapy; (2) postural drainage and other ancillary supportive measures; (3) recognition and amelioration of coexisting medical and psychologic diseases; (4) marginal minor surgical procedures; and (5) early recognition of cases in which major surgery is indicated at the outset.

ANTIBIOTIC THERAPY

Sulfonamides. The sulfonamides, particularly sulfadiazine, altered the therapeutic approach somewhat, but because of their relatively narrow antimicrobial spectrum they produced no astonishing results.[12,13]

Penicillin. From the first studies with penicillin it was soon apparent that this was an invaluable drug in the treatment of pulmonary suppuration.[14] It still remains as the mainstay of antibiotic therapy in acute lung abscess. Its particular efficacy no doubt relates to the predominantly gram-positive microorganisms and oral spirochetes of the oropharyngeal flora which are the chief primary pathogens responsible for the average case of acute lung abscess. We believe that, preferably but not obligatorily, with complementary sulfadiazine it will produce as satisfactory a result in acute lung abscess as any other single antibiotic. The following principles must be heeded in treating acute lung abscess with penicillin: (1) the pathogens must be identified and known to be sensitive; (2) early and vigorous treatment is indicated, with doses significantly exceeding those commonly used for the majority of other infectious diseases; (3) treatment must be sufficiently prolonged since the cure time of lung abscess is longer than that encountered with other pyogenic infections; and (4) better results are obtained with aerobic than with anaerobic abscesses. The dosage schedule we employ at the Harlem Hospital is 600,000 units of procaine-penicillin G intramuscularly every six hours until the patient is clinically well, then twice daily until there is complete radiologic resolution of the lesion with only residual minor irreversible fibrotic changes. When sulfadiazine is used in combination with penicillin, we use a dose of 1 gm. every four hours.

Penicillin G diethylaminoethyl ester hydriodide (neo-penil®) has been recommended in the treatment of pulmonary infections sus-

ceptible to penicillin, since greater lung tissue and sputum levels are reached with this compound than with penicillin salts, including procaine penicillin. We have not used this preparation at the Harlem Hospital and are not aware of any controlled studies in acute lung abscess which have demonstrated the unequivocal superiority of this ester over procaine-penicillin. By the same token a definite role in the treatment of acute lung abscess has not been shown for dibenzyl-ethylenediamine dipenicillin G (bicillin®), which produces a two- to four-week therapeutic blood level.

We have not encountered any hypersensitivity reactions to parenteral penicillin in any of our cases. In view of the known increased frequency of such reaction, however, we are aware that therapeutic concepts may change in the near future, at least to the extent of obligatory sensitivity tests before initiating long-term penicillin therapy.

Streptomycin and Dihydrostreptomycin. These drugs, single and in combination, have done much to solve the problem of gram-negative organisms immune to all forms of penicillin. Many clinicians have substituted a penicillin-streptomycin regimen for penicillin and sulfadiazine. Harter considers 100,000 units of penicillin every three hours and ½ gm. of streptomycin every twelve hours a satisfactory regimen for the treatment of acute lung abscess.[4] We have not used this combination at the Harlem Hospital because of the danger of masking a coexisting tuberculous infection. Eighth cranial nerve involvement following prolonged streptomycin or dihydrostreptomycin therapy is always a real possibility and should be preceded by audiometric tests.

Chloramphenicol (Chloromycetin®). Although this drug possesses a wide antimicrobial range, its potential danger of inducing blood dyscrasia interdicts its prolonged use without careful laboratory control. It has been used successfully in chronic pulmonary suppuration.[23] We have not used it in our cases.

Tetracycline Derivatives. Chlortetracycline (aureomycin) and oxytetracycline (terramycin). While these drugs have certain marked advantages, chiefly their incredibly broad range of antimicrobial activity and the ease of administration inherent in the oral route of medication, we have failed to note any dramatic response to either of them. In a number of our cases we were successful in effecting a cure of an acute abscess with the cycline drugs in cases in which penicillin had apparently failed, but we believe it is not beyond the range of possibility that penicillin had made the job easier for the results later obtained with the tetracycline derivatives. The obvious disadvantages of these drugs are their greater cost, the danger of emergence of pathogenic yeasts due to excessively thorough respiratory bacterial elimination, and the occurrence of tocopherol (vitamin K) deficiencies secondary to intestinal flora destruction. The latter is particularly significant, as abscess cavities, particularly the putrid variety, are prone to bleed easily even in the absence of lowered blood vitamin K levels.

We have used both aureomycin and terramycin in dosages of 250 mg. every six hours until clinical cure is attained, then three times daily until complete radiologic clearing is seen. In two patients admitted in an advanced state of sepsis, we administered initial doses of 500 mg. of terramycin in 5 per cent glucose intravenously concurrently with oral medication. We believe this procedure to have been life-saving in these two cases.

All things considered, it is believed best to reserve the tetracycline series for unusually stubborn cases in which penicillin has failed after an adequate trial period, or in those cases in which the causative organisms are demonstrated to be generically or individually immune to penicillin and for those cases which are extremely toxic. An alternate approach in treatment would be the development of a basic penicillin regimen interrupted by brief periods of tetracycline derivative therapy, as suggested by Barach.[15] This method ingeniously harnesses the natural antagonism of the bacterial groups, first allowing the gram-negative organisms to aid in the elimination of their gram-positive antagonists already belabored by penicillin, and then conquering the conquerors by the tetracyclines.

Tetracycline (Tetracyn,® Achromycin®) and Erythromycin.® These drugs are relative newcomers on the antibiotic scene and have not yet been properly evaluated for their specific activity in acute lung abscess. Theoretically, at least, they should be of considerable usefulness in gram-positive coccal infections, particularly where hypersensitivity to other antibiotics exists. Erythromycin is said to alter the intes-

tinal flora to a lesser extent than the other wide-spectrum antibiotics.

POSTURAL DRAINAGE AND OTHER ANCILLARY MEASURES

Zeal over pharmacotherapy often obscures the need for the rigid enforcement of postural drainage, supportive therapy, good nursing care and such other special procedures as common sense will dictate. There is little excuse for not instituting prompt postural drainage. The optimal position can easily be determined from precise knowledge of bronchopulmonary segmental anatomy and the specialized radiologic technics, as tomography, which we are now able to employ in determining the exact location of a pulmonary lesion. The duration and frequency of drainage must be decided on an individual basis and will depend on such factors as type of patient, coexisting diseases, facilities and convenience of the drainage position. Massages or the use of vibratory apparatus over the abscess site will often serve to loosen thick mucus plugs. Inhalation of tryptar® (Armour brand of trypsin) aerosol by nebulizer or aerosol mask, may liquefy tenacious sputum. Gentle coughing exercises should also be taught to the patient. Parenteral and oral vitamin therapy, liver, iron and other hemopoietics, high-protein high-calorie diets, careful attention to a well functioning alimentary tract, and prompt alleviation of distressing symptoms by palliative medication are all measures to be observed in a medical regimen. Unfortunately, however, these measures are often left in the hands of a harassed nursing staff, taken for granted or inadequately stressed. Repeated small transfusions of 250 to 300 cc. of whole blood are often helpful in regaining lost ground, especially when a severe anemia has supervened. In acute stages oxygen should be given by catheter, mask or tent to relieve respiratory embarrassment.

The Chronic Chest Disease Service of the Harlem Hospital has every patient with acute lung abscess placed on a "supportive regimen" consisting of multivitamins, feosol and protein supplements daily. In severe cases liver and iron is administered intramuscularly and 1000 cc. of amigen solution intravenously daily. If the erythrocyte count is less than 3 million, transfusions are added in the early stages of the disease to correct promptly the acute anemia which is a common finding in cases with overwhelming toxicity.

COEXISTING MEDICAL AND PSYCHOLOGIC DISEASES

A substantial number of patients with acute lung abscess admitted to hospitals unfortunately come from beyond the pale of accepted social standards. In this category are met the underprivileged, undernourished, drug and alcohol addicted, and mentally subaverage. Such people are frequently desperate, have poor insight and show impaired judgment. The rate at which such patients leave the hospital against advice is high. Of 105 cases of lung abscess admitted to the Harlem Hospital from April 1, 1949, to April, 1954, twenty left the hospital against advice as soon as there was some degree of clinical improvement. Many of those who did remain to completion of therapy were periodic behavior problems. In dealing with these patients, therefore, the greatest amount of tact, patience and understanding is required. If facilities for psychiatric guidance are available, they should be promptly applied. Since the average case of acute lung abscess will be hospitalized for approximately two months, some form of recreation or occupational therapy must be provided to maintain morale and optimal patient cooperation.

Coexisting medical diseases, both predisposing and unrelated to the abscess, are extremely common. Malnutrition, cardiac disease, neurologic disorders, chiefly epilepsy, thrombophlebitis and alcoholic syndromes including frank delirium tremens are the most common. Of the twenty-four deaths we have had in our series, twenty occurred in the presence of such diseases and four were due to overwhelming septicemia. It is clear that a thorough medical evaluation in every case of acute lung abscess is mandatory and immediate therapy should be instituted for any recognized predisposing, intercurrent or complicating disease which may exist.

SUMMARY OF 105 CASES WITH SUPPURATIVE LUNG DISEASE

In twenty cases the pulmonary disease was secondary to some other fatal systemic disease. In eight cases the lung disease was chronic and presented complications necessitating surgical management on admission. Four cases of primary lung abscess were complicated by

overwhelming septicemia beyond help by medical treatment. Seventy-three cases of acute uncomplicated lung abscess were treated with antibiotics. Of these twenty left the hospital against medical advice after being clinically improved but not cured. Fifty-three patients completed treatment and were cured medically. Treatment in these cases was as follows: terramycin, fourteen cases; aureomycin, thirteen; penicillin and sulfadiazine, ten; penicillin, three; penicillin, then aureomycin, four; penicillin, then terramycin, seven; streptomycin and terramycin combined, one; and penicillin, then aureomycin and terramycin, 1.

Before discharge from the hospital those patients who have been successfully treated should be instructed to avoid situations which were shown to have caused or precipitated the abscess. A well organized social service can be of inestimable value here. Periodic clinic follow-up care is also essential after discharge from the hospital.

MARGINAL MINOR SURGICAL PROCEDURES

In the field of chest diseases, as in every other comparable field, there is an overlapping area between pure medicine and definite surgery. The dividing line will differ with individuals and institutions. Some of the procedures in this twilight zone are chest taps, pneumothorax, enzymatic débridement and topical treatment. The necessity of these measures will arise only when a relatively minor complication has occurred or a major one is threatening.

Hemoptysis. Minimal hemoptysis may be controlled by sedation and intravenous tocopherol, calcium, procaine or pituitrin, or any rational combination of these. Artifical pneumothorax and other forms of collapse and immobilization therapy are occasionally advocated for the closure of cavities responsible for massive hemorrhage, but in general these measures have fallen into deserved disrepute because of the interference with free drainage which they produce.

Empyema. In loculated types of empyema complicating peripherally situated abscesses in which simple aspiration has proved to be of limited value, enzymatic débridement by the intrapleural instillation of trypsin, streptokinase, streptodornase or varidase® (streptokinase-streptodornase, Lederle), may circumvent the necessity of external drainage. In cases of pleural effusion, whether suppurative or not, aspiration should be followed by the intrapleural instillation of the antibiotics. In the Harlem Hospital we routinely use 1 million units of aqueous penicillin and 5 gm. of streptomycin in 100 cc. of physiologic saline. We have not encountered a hyperergic reaction, but since this is possible, especially when the enzymes are used, we routinely antihistaminize patients before instilling antibiotics or enzymes into the pleural cavity.

Pyopneumotborax. Where pyopneumothorax is associated with a marked mediastinal shift and acute respiratory distress, underwater drainage may be a life-saving measure. In more severe cases, or where a ball-valve mechanism is present, the involved hemithorax may have to be kept under continuous negative pressure.

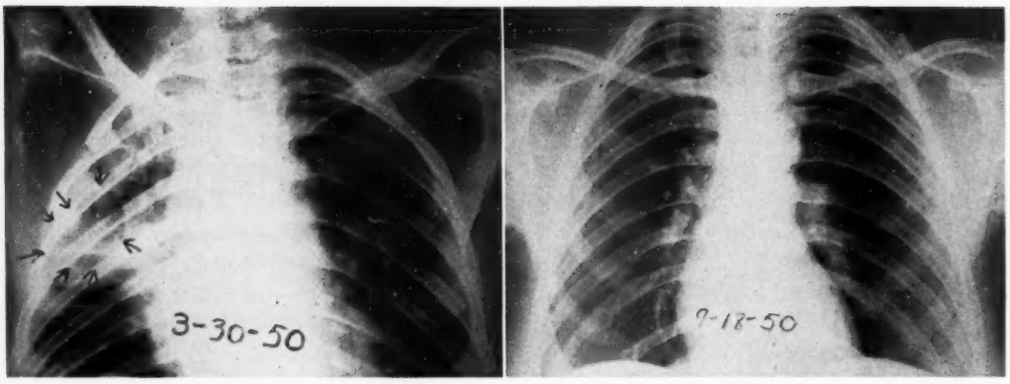
Aerosol Therapy. Aerosol therapy with the antibiotics, particularly penicillin, alone or in supplementation of oral or intramuscular modalities, has met with much praise.[8,17] Eastlake believes that extirpation of even chronic abscesses may be avoided in many cases by aerosolized solutions of penicillin for at least one month after maximum benefit has been obtained from surgical drainage.[18]

Topical Treatment. Topical treatment of lung abscess has been described by Burnett et al.[19] whereby aspiration is performed under fluoroscopic guidance followed by the instillation of 100,000 units of penicillin and 1 gm. of streptomycin in 1 to 5 cc. of a wetting agent. This treatment is supplemented by aerosol inhalations four times daily. These authors stress, however, that this procedure should be attempted only by a qualified thoracic surgeon.

SELECTION OF CASES FOR MAJOR SURGERY

It is not within the scope of this paper to evaluate the various surgical procedures used formerly or currently in the treatment of acute lung abscess. Reference to this information may be found elsewhere in the literature.[20] Nevertheless a statement of a few principles which have guided us in the selection of our cases is in order.

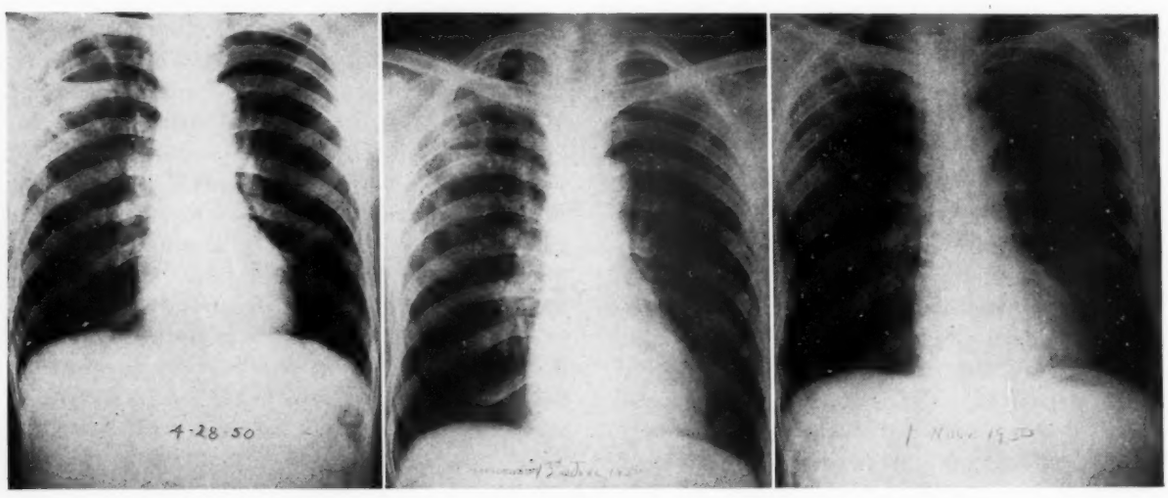
Bronchoscopy. Bronchoscopy should always be employed wherever the etiology of an acute abscess is obscure, where the response to conservative therapy is slow, or where a foreign body or malignant growth is suspected. The value of a bronchoscopy is both diagnostic and therapeutic. Bronchial washings obtained



1 2

FIG. 1. Shows a cavitaric lesion with fluid level in a toxic, extremely ill patient. The abscess involved the entire right upper lobe.

FIG. 2. About six months later shows complete healing with minimal fibrotic scarring in the right infraclavicular zone.



3 4 5

FIG. 3. Shows an abscess in the dorsal segment of the right upper lobe. There is a cavity with a fluid level.

FIG. 4. Six weeks later shows a marked reduction in the size of the lesion and of the cavity which still contains a fluid level.

FIG. 5. Taken several months after discharge shows the healed right upper lobe lesion with fibrotic changes in the right infraclavicular zone.

by this procedure may be studied bacteriologically and cytologically, tissue biopsies may be obtained from suspicious areas and foreign bodies removed if present. Bronchial drainage is facilitated and recovery accelerated. Intrabronchial instillation of streptokinase and streptodornase has been described by Miller, Surmonte, and Long.[24] Rubin, Goldstein, and Rubin believe that bronchoscopy should never be omitted, and that every patient should have at least one bronchoscopy in the course of treatment.[8] We do not have our cases bronchoscoped routinely but insist on same whenever any of the foregoing indications exist.

External Drainage. In our view this should never be the surgical treatment of choice. We reserve recommendation of this procedure only where a definite contraindication to excisional surgery exists. In those cases, however, in which external drainage is done, it should never be performed through normal pulmonary tissue by way of artificially produced adhesions because of the danger of spread to normal collateral areas of lung unprotected by a defen-

sive inflammatory reaction. Moreover metastatic brain abscesses may follow since the venous channels are not closed by protective inflammatory thrombosis. The pointing of the abscess must therefore be precisely localized and the drainage established through this site.[21] If, on entering the thorax, it is seen that this site has been missed, it is best to close the wound and re-enter the thorax at the proper site.[5]

Resectional Surgery. It is imperative that resection should remove the diseased area completely with a minimum sacrifice of healthy lung tissue.[22] This is vital not only for immediate functional results but also because of the possibility of a later indication for resection for an unallied condition. It is incumbent both on the surgeon and internist to determine exactly what the extent of the disease is. Resection should be limited to lobectomy or preferably segmental resection.

Pneumonectomy. This radical step is rarely indicated for lung abscess. It should be reserved only for those cases in which fairly good evidence exists that the underlying process is neoplastic, in which extensive fibrosis has caused such mediastinal shifting that there is marked interference with cardiovascular dynamics, or in which the diseased lung has undergone advanced damage while the pulmonary circulation has remained intact, resulting in negligible oxygen intake in the diseased lung. The latter condition produces a functional arteriovenous fistula and is demonstrable by proper pulmonary function tests.

Even when a case has been earmarked for surgery, whether from the very onset or at any time in its course, sound medical management as indicated elsewhere in this paper must apply in order to render the patient an optimal risk for surgery.

In our last published paper on acute lung abscess,[3] we reported a series of thirty-seven successfully treated patients. Of these, ten were treated with penicillin, three with penicillin and sulfadiazine, twelve with aureomycin, six with terramycin, four started on penicillin and completed with aureomycin, and two started on penicillin and completed with terramycin. We were unable to conclude that any single antibiotic or combination of antibiotics was distinctly superior to the others.

Since that published series we have successfully treated sixteen more patients; eight with terramycin, one with aureomycin, one with combined streptomycin and terramycin, one started on penicillin and completed with aureomycin and terramycin, and five started on penicillin and completed with terramycin. We have deliberately varied the therapy in a persistent search for an optimal combination of antibiotics, but are still unable to deviate from our previously held conclusions that none of these drugs holds a clear superiority over the others, but that successful results depend on prompt and adequate treatment. The clinical cure time still remains about twenty days and total cure time (clinical plus radiologic) about two months. Our only adverse criticism with regard to penicillin is the necessity for its intramuscular administration. This, however, is greatly outweighed by its negligible cost.

The accompanying reproductions of chest x-rays of two cases are typical of our results with medical management. Figures 1 to 5 represent the radiologic findings and results in the fifty-three cases of uncomplicated acute lung abscess which we treated and cured medically between the period April, 1949, and April, 1954.

CONCLUSIONS

Our experience in the treatment of suppurative disease of the lung leads us to be of the opinion that acute uncomplicated lung abscess is at present a medical problem. Also, that the role of surgery in chronic cases and those early cases with complications must be clearly recognized; in this type of case the joint efforts of the internist and the surgeon as a team provides the best results. Further, that the number of patients requiring surgery will become increasingly smaller if prompt and adequate antibiotic and ancillary medical measures are instituted early in the course of acute suppurative disease of a primary nature.

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Lung Abscess—A Medicosurgical Problem

DAVID H. WATERMAN, M.D., SHELDON E. DOMM, M.D. AND WILLIAM K. ROGERS, M.D.,
Knoxville, Tennessee

From the Thoracic Surgical Services of Fort Sanders Presbyterian Hospital and Knoxville General Hospital, Knoxville, Tenn.

THREE fallacies concerning lung abscess would seem to be currently present in the minds of many physicians. The first of these is that lung abscess has become so rare a disease that it is no longer of importance. The second is that medical treatment alone suffices in practically all of the few cases that are encountered. The third is that resection is the only surgical procedure to be considered in the remaining cases. Actually there is some basis of fact for each of these concepts; they are erroneous simply because they are carried too far.

Regarding the first of these, it must be remembered that the etiologic factors producing lung abscess, such as dental sepsis, aspiration of vomitus or other infective material, and various types of bronchial block have not been eliminated by antibiotics. Earlier treatment of the resulting pulmonary infections with the antibiotics, however, and the wider acceptance of prompt bronchoscopy certainly should result in fewer abscesses. Nevertheless a review of our own figures shows we are still encountering as many cases of lung abscess and suppurative pneumonia as we did in the early forties. For example, in the past two years we have treated over fifty cases. In a recent article Gittens and Mihaly[1] have reported thirty-seven abscesses in the acute uncomplicated category alone at the Harlem Hospital from April, 1949 to April, 1953. Figures from other thoracic clinics give further evidence that the problem has by no means been eliminated.

That the wide-spectrum antibiotics have mitigated the severity of lung abscess and suppurative pneumonia is undeniably true. The highly toxic, desperately ill lung abscess patient is now rarely rather than commonly seen. The entire therapy picture is materially brighter than the complicated one of a relatively few years ago when the frequently quoted figures of Allen and Blackman,[2] and Smith[3] showed over 34 per cent mortality in a combined collective series of 4,280 cases.

In 1952 the authors[4] analyzed 218 cases of lung abscess and suppurative pneumonia personally treated. Of these, 134 cases were treated by conservative measures consisting of bronchoscopy and antibiotics. Good results were obtained in 77 per cent of the seventy-four seen in the postantibiotic era, in contrast to 63.3 per cent of the sixty seen in the preantibiotic period. Significantly there was a marked drop in mortality from 23.3 per cent in the preantibiotic era to 4 per cent in the postantibiotic. It should be pointed out that these are completely unselected figures and represent all cases which were treated, whether acute, subacute or chronic.

On the other hand in highly selected series, such as those reported by Gittens and Mihaly in 1954[1] in which nothing but simple uncomplicated abscesses seen early in the course of the disease were included, the results were uniformly excellent without surgery. Stivelman and Kavee[5] presented a similarly optimistic picture in 1949. While our own experience in the majority of such cases closely parallels this, it must be borne in mind that in actual practice not all abscesses are seen early in the acute stage, nor are these responsive to medical treatment alone in all cases. We have had occasion to observe several acute abscesses in the past few years that were rapidly fulminating in spite of massive combined antibiotic therapy. We are also encountering abscesses presenting a bacterial flora resistant to almost all antibiotics.

While our over-all mortality since antibiotics have been used has been gratifyingly low, we have actually seen no decrease but rather an increase in the number of cases coming to surgery. Probably this is due to the effect of antibiotics in relieving symptoms and obscuring diagnosis so that more abscesses progress to the chronic stage. Once an abscess

is chronic or complicated, with permanent parenchymal and bronchial damage resulting, the golden opportunity for medical therapy is already passed.

As to the third fallacy, the published figures on resection of chronic abscess, such as those

TABLE 1

Lesion	No. of Cases	Per cent
1. Lung abscess and suppurative pneumonia	268	57.8
2. Carcinoma with secondary suppuration	187	40.3
3. Adenoma with secondary suppuration	3	0.6
4. Embolic abscess with pyemia	6	1.3
Totals	464	100.0

of Shaw and Paulson[6] with only 3.8 per cent mortality in fifty-two cases, have been so favorable that many physicians have assumed that no place remains for other types of surgical therapy. Admittedly the indications for open drainage are markedly narrowed by the efficacy of conservative therapy on the one hand, and the improved mortality of resection on the other. Most recent surgical contributors, however, such as Byron[7] and Cowley,[8] utilize surgical drainage not infrequently. Bad risk patients, for example, constitute a rather sizable group and frequently can be salvaged by this procedure. Physicians should not have to be reminded that surgical drainage of suppuration is still a valid principle.

MEDICOSURGICAL MANAGEMENT

Numerous surgical writers, such as Glover and Clagett,[9] and Klepser and Davis,[10] have expressed the viewpoint that lung abscess is a surgical problem from the outset. While we entirely subscribe to such a dictum in the strict sense, it is perhaps more politic to state that lung abscess is a medicosurgical problem.

From 1941 to the present time 464 cases of abscess or suppurative pneumonia have come under our cognizance. This number does not include cases of mycotic infection, bronchiectasis or tuberculosis, but does include those cases associated with malignant disease in order to show the increasing role carcinoma plays as an etiologic agent. Table 1 is the breakdown of these cases and shows that carcinoma with secondary suppuration contributes 40.3 per cent to the total.

With bronchogenic carcinoma on the increase it can be predicted that an even greater incidence will be encountered. Thus it would seem mandatory that every case of lung abscess, whether acute or chronic, be carefully investigated for malignant background, a viewpoint shared by Brock,[11] and Strang and Simpson.[12]

It has been our good fortune to be able to apply either conservative or surgical therapy as we saw fit in most of the cases in our series. In a high percentage of these it has not been necessary to resort to surgical measures other than bronchoscopy. Therefore we do not believe that our figures are weighted in favor of either medical or surgical treatment.

It is the purpose of this report to outline the regimen that has proved the most efficacious in our hands in treating the remaining 268 cases listed in the first line of Table 1. Roughly 70 per cent of these were putrid and 30 per cent nonputrid. For the purposes of discussion the steps in management are listed under the following headings.

1. *Preliminary Work-up.* The prompt evaluation of each patient after he is admitted to the hospital will, in the majority of instances, permit application of rational therapy from the outset. The importance of a careful history, bearing in mind the possibility of bronchogenic carcinoma as an etiologic agent, need not be emphasized. Physical examination must be thorough. Six-foot erect postero-anterior and lateral x-rays will give the exact size and segmental location of the lesion. Almost one-third of these cases will not show the characteristic fluid level due to bronchial block. In addition to the routine laboratory work a packed cell volume determination may be of advantage. The sputum is examined on admission for acid-fast bacilli, but cultures and smears for other organisms are not done at this time in view of the well known contamination by mouth organisms, and the consequent superiority of a bronchoscopic specimen. The appearance and odor of the sputum are noted—the use of a glass container permits ready inspection—and the volume is recorded daily. Day and night temperatures are kept of course.

Routine administration of antibiotics is not begun on admission except in the rare instance when extreme toxicity makes this imperative.

2. *Bronchoscopy.* In our opinion every case of lung abscess or suppurative pneumonia demands bronchoscopy within the first twenty-four hours. The procedure is so simple and so fruitful that it is difficult to see why any physician should hesitate to apply it promptly, or why some writers would seem to show a lack of enthusiasm for the procedure. We have never seen a patient with lung abscess or suppurative pneumonia who was too ill to be bronchoscoped, although we have seen many too ill to permit any delay in performing the procedure.

From a diagnostic standpoint the possibility of malignancy makes its use imperative, inasmuch as most cases of malignancy accompanied by lung abscess are sufficiently advanced to permit diagnosis via the bronchoscope. In addition to ruling out other pathologic disorder, valuable information is gained regarding the suppurative process, in that the inflammatory reaction can be localized and correlated with the x-ray findings and the bronchial reaction and block factors evaluated. Fox, Hughes, and Sutliff[13] also emphasize that diagnostic bronchoscopy is essential.

A second important function of bronchoscopy, related to the first, is the collection of bronchial secretions uncontaminated by mouth organisms for laboratory study. In addition to the routine smears, aerobic and anaerobic cultures, tuberculosis smears and cultures, and fungus cultures, it has been our practice in all of our abscesses to order sensitivity studies for antibiotics because of the disturbing frequency with which we are encountering cases presenting a bacterial flora resistant to almost all the available chemotherapeutic agents. This resistance may not necessarily correlate with previous antibiotic administration in the individual. In recent months we have found a surprising number of infections sensitive only to chloromycetin,® and have postulated that this may be due to the more restricted use of this agent by the average physician following the reports of complications after prolonged administration. As Hewitt[14] has pointed out, the bacteriostatic and bacteriocidal specificity of the antibiotics has made clear the necessity for more, rather than less, specific bacteriologic diagnosis if proper treatment is to be applied.

From a therapeutic standpoint bronchoscopy has long been advocated as an essential prerequisite to effective conservative management. An infection in the lung is like any other infection in that it must drain adequately if it is to clear. A competently performed bronchoscopy with thorough systematic aspiration of all branch bronchi, particularly in the affected segments, will promote bronchial drainage as will no other measure. Anyone who has repeatedly observed via the bronchoscope the typical angry, swollen bronchial mucous membrane can well understand how this bronchial drainage cannot be achieved without mechanical help. An efficient bronchoscopy evacuates accumulated and obstructing bronchial secretions (even to the extent of actually mechanically opening the lung abscess in some cases), permits shrinking of the mucous membrane by topical application, thus relieving further block, and produces selective cough that makes continued effective raising of secretions possible.

The favorable effect of a bronchoscopy is usually manifested within the first few days or even the first few hours, and may be very dramatic. As a rule the patient begins to raise secretions effectively in large amounts, often when little was produced before. The odor of the sputum may even disappear overnight, as the anaerobes diminish with effective drainage of the abscess. Cough is increased the first day or two following the bronchoscopy, if it is well performed, but diminishes as the sputum volume begins to decrease. The temperature usually falls, sometimes precipitously, and the patient exhibits evidence of reduced toxicity and increased well-being. Bronchoscopy need not be repeated in the average case more often than every week or ten days, as the beneficial effect should last about this period of time. In our opinion more frequent bronchoscopy is rarely necessary and frequently is harmful. Often only a single bronchoscopy is necessary, but if the salutary effects are not observed fairly promptly, the patient is rebronchoscoped several days later. If improvement does not ensue following the second bronchoscopy, it is evident that a change in therapy should be considered. The bronchoscope should be recognized simply as a tool that can be used by the thoracic surgeon to contribute an important phase of therapy in lung abscess. Bronchoscopy is not a definitive treatment in itself.

Various authors have advocated the instilla-

tion of antibiotic agents directly through the bronchoscope, but our results with aerosol antibiotics have been so satisfactory that we have resorted to direct instillation only infrequently.

3. *Postural Drainage.* Immediately following the bronchoscopy the patient is placed on a postural drainage regimen four times a day, or more often if there are profuse secretions. It is our practice to use an expectorant such as enteric-coated ammonium chloride, 22½ gr., followed by a glass of hot water one-half hour before meals and before bedtime. The patient then takes his postural drainage and coughs vigorously in the position for two or three minutes. It must be emphasized that postural drainage should be individualized, as incorrect postural drainage may do more harm than good. Postural drainage by definition is that position which most effectively aids drainage. In a middle lobe for example, the patient should lie flat on his back. In lower lobes the position is well down over the bed as near vertically as possible. In upper lobes the patient may either lie on his face or his back or sit upright, depending upon the segment involved. After the individualized position has brought the secretion to the main bronchus, the head-down position may be used briefly to advantage. Experimentation will find the most effective position or combination of positions. This routine should then be carefully demonstrated and supervised so that it may be utilized effectively. Postural drainage should be continued as long as the abscess persists, even if the patient's sputum drops to an almost negligible amount. The dryer the patient's lung can be kept, the quicker will be his recovery.

4. *Antibiotics.* Once bronchoscopy, making possible bronchial drainage, has been done and the bronchial secretions obtained for laboratory study, the vigorous administration of antibiotics is begun. Until the laboratory studies provide accurate direction to antibiotic therapy, the most satisfactory agent for immediate use is penicillin. This has been the most effective antibiotic in our hands, and we agree with Hewitt that the aqueous preparations of penicillin, such as penicillin G potassium, are superior to the repository ones during the initial phase of therapy. Lung abscess is potentially highly dangerous, and penicillin in bacteriocidal doses is warranted. We administer 100,000 to 300,000 units every three hours, using the hypoallergic preparations if indicated. Frequently we use the procaine penicillin at bedtime, however, so that the patient may have uninterrupted rest.

Penicillin is also administered by the aerosol route in doses of 25,000 to 50,000 units every three hours during the day for three or four days following the bronchoscopy. A bronchorelaxing drug, such as neo-synephrine,® vaponefrin,® isuprel,® or prothricin,® providing a shrinking agent plus tyrothricin, is added to each inhalation. This usually results in rapid improvement in the severe inflammatory bronchial reaction so typical of lung abscess. We have now had a wide experience in the use of such aerosol antibiotics after bronchoscopy in some 4,000 cases of bronchial and pulmonary infection and have been particularly enthusiastic about the results in our lung abscess group. We have found the penicillin aerosol much superior to penicillin dust. Terramycin® is the only other antibiotic we have found advantageous by the aerosol route. Trypsin inhalations, however, may aid greatly in liquifying secretions.

As soon as the sensitivity studies are reported, usually within the first few days of treatment, the antibiotic therapy is modified as indicated. Actually the use of sensitivity studies has simplified rather than complicated the treatment of infection, as the antibiotic of maximum effectiveness can thus be chosen without experimentation.

The physician should be constantly aware that the antibiotic initially used may so change the bacterial flora as to become ineffective. This is usually manifested by a reversal in response to therapy or by an actual increase of the suppurative process on x-ray. When such change occurs we usually rebronchoscope the patient and obtain additional bacteriologic cultures and sensitivity studies. Fortunately a large number of antibiotics are now available for alternative therapy.

Should the patient develop sudden dyspnea and chest pain, the physician must be alert to the possibility of rupture of the abscess into the pleural cavity and should drain the resulting empyema as an emergency procedure. Not infrequently the abscess may go on to healing once the pleural cavity drainage is provided.

5. *X-rays.* Weekly x-rays in the erect postero-anterior and lateral positions are the best guide to the real progress of the case.

Unless there is definite x-ray improvement at weekly intervals, surgical intervention should be considered, granted that bronchoscopy and a change in antibiotic therapy are ineffectual. A stationary course is actually a retrogressive one in lung abscess, and procrastination may cost the life of the patient.

If the abscess is a chronic one and surgical resection is believed indicated, bronchography can be of great value in delineating the extent of the resection. Frequently minor bronchiectatic changes are present adjacent to the abscess which might be missed were the bronchogram not taken. No deleterious effects of bronchography have been noted in our series.

6. *Open Drainage.* Three indications still are present for open drainage. The first of these is the occasional fulminating acute abscess that fails to respond to conservative treatment with bronchoscopy and antibiotics and which must be drained as a life-saving measure. These are rare cases, but are just as much a problem in therapy as they were in the preantibiotic era.

The second indication is in the patient either too old or too ill to be safely resected. Attendant heart disease, for example, or another serious lesion elsewhere may make the patient too severe a risk to undergo extirpation surgery. Occasionally the relatively easy procedure of open drainage may be more practical in civilian or military situations in which the experience of the surgeon, the available hospital or anesthesia facilities render resection a hazardous undertaking.

The third indication is the solitary uncomplicated acute abscess peripherally located in which a good result from drainage can be anticipated, even though resection surgery would be entirely feasible from a risk standpoint. Open drainage is a simple procedure and is less risky than major thoracotomy. Neuhof, Touroff and their associates[15–19] demonstrated before our present antibiotics were available that open drainage carried only a 2.43 per cent mortality in a large series of acute lung abscesses. With antibiotics this should be even less.

It should also be pointed out that resection implies loss of lung tissue, whereas open drainage may conserve it. Since the majority of acute cases will respond to conservative therapy, however, there will be few cases encountered, even at best, in which open drainage is desirable. It has long been recognized that chronic abscess is rarely suitable for drainage, except as a palliative procedure, and is always an indication for resection in the reasonable risk patient. Generally speaking, good results in open drainage will be in direct proportion to the age of the abscess. It might also be mentioned that open drainage does not preclude later resection.

As to technic, a single stage drainage procedure under local anesthesia can almost always be applied. If pleural symphysis is not present, the visceral pleura may be sutured to the parietal before the latter is open. We have done this for a number of years without encountering the complication of empyema. In the majority of cases, however, the cone of adhesions present is adequate. If the abscess has been accurately localized on 6-foot postero-anterior and lateral films, it is possible to resect a small piece of rib directly over the abscess and enter it with little difficulty. We believe that abscesses heal concentrically, and hence place the tube in the center rather than at the bottom of the abscess. The abscess is packed with washed iodoform gauze brought out through the large rubber drainage tube, and repacked daily until all slough is removed and the abscess cavity reduces in size satisfactorily. The tube is not removed until the abscess is completely obliterated. Cultures taken directly from the abscess guide therapy if an antibiotic is indicated. Routine biopsy of the abscess wall may reveal unsuspected carcinoma or tuberculosis. With the protection afforded by antibiotics at the present time, it is frequently possible to discharge the patient from the hospital soon after operation and continue dressings at the surgeon's office. This may effect considerable financial savings for the patient.

7. *Resection Therapy.* Once an abscess has become complicated or has persisted over a period of several weeks beyond the acute stage, permanent pulmonary damage results and resection therapy is indicated. The most signal advance in recent years has been the reduction in surgical mortality in resection from the previous appallingly high figure of the preantibiotic era to a respectable one of below 5 per cent in most series. In our own experience of fifty-one cases resected since the advent of antibiotics the mortality has been 1.9 per cent. This is in sharp contrast to our 28.6 per cent mortality before antibiotics were used. The safety with which resection therapy

can now be performed under antibiotic coverage has made it possible to salvage a high percentage of patients who once remained either chronic or who succumbed to their disease.

Cowley[8] has mentioned brisk hemorrhage as an indication for surgical intervention. In our own experience we have not had occasion to resort to resection in such an instance, but would not hesitate to do so if the need should arise.

It has been our custom to prepare these patients for resection with intensive intramuscular penicillin for two or three days before operation, and with aerosol penicillin prescribed concurrently to temporarily sterilize the bronchial tree. If the sensitivity studies have indicated that another antibiotic is more effective, it is of course substituted. As mentioned previously, most of these patients who come to surgery have already had bronchography so definitive surgery can be well planned.

Individual ligation technic is superior to wedge resection in most cases of lung abscess. In order to preserve all lung tissue possible, segmental resection is applied whenever possible. In spite of the fact that air leaks are more common with segmental technic, we have found it possible to obliterate the residual space rapidly with vigorous active suction and thus avoid the complication of empyema. Empyema is also relatively rare when the entire lobe is removed, inasmuch as the remaining lobe or lobes can be left with few air leaks, and speedily expand to fill the space.

Empyema remains a grave problem particularly in pneumonectomy. We have had an incidence of almost 50 per cent, as have Glover and Clagett.[9] Because of this high figure we strongly believe that a relaxing type of thoracoplasty should be performed rather promptly following the pneumonectomy. To prevent scoliosis the first rib is usually left in place. Once bronchopleural fistulas and empyema have developed, however, the situation is enormously complicated, as multiple stage thoracoplasties may fail to obliterate the space and close the fistulas.

In 1950 Holman[20] reported a technic whereby chronic lung abscesses could be peeled out of the parenchyma without sacrificing the entire lobe or segment. As in a previous report, we continue to be interested in this method of resection and consider it a meritorious contribution. Those surgeons who have had the experience of enucleating tuberculous nodules from the lung will find the technic equally applicable in abscess.

8. *Final Evaluation.* A lung abscess case should be followed carefully, whether his treatment be conservative or surgical, until it is certain no residual damage remains and the original lung abscess cavity has been completely obliterated. We believe that it is mandatory that a bronchogram be taken in all cases of conservative or drainage therapy. Touroff, Nabatoff and Neuhof,[21] by carefully evaluating their large drainage series, found 13.4 per cent had some residual change. Our experience has been comparable. When appreciable parenchymal or bronchial damage has been demonstrated by the bronchogram, elective resection is considered.

SUMMARY AND CONCLUSIONS

1. Three fallacies currently persist regarding lung abscess: (a) that it is now so rare as to be unimportant, (b) that antibiotics alone suffice as therapy and (c) that resection is the only surgical measure of value.

2. Bronchogenic carcinoma is an increasingly common cause of lung abscess and should be considered as the etiologic agent in every case. Forty per cent of a total of 464 cases were malignant.

3. Based on the authors' experience with 268 cases of lung abscess and suppurative pneumonia of non-malignant etiology, a regimen of medicosurgical management is outlined.

4. A careful preliminary work-up serves as a guide for rational therapy.

5. Bronchoscopy is indispensable in lung abscess and should be applied early, both for diagnosis and therapy. Bronchial secretions obtained at bronchoscopy provide accurate and essential bacteriologic data. The limitations of bronchoscopy must be recognized, inasmuch as it is not a definitive treatment.

6. Postural drainage, correctly applied, is an essential supplement to bronchoscopy in conservative therapy.

7. Antibiotics have changed the entire picture for the better. They should be applied vigorously but intelligently under bacteriologic guidance. Sensitivity studies should be routine. Penicillin remains the most effective antibiotic, both parenterally and by the aerosol route.

8. Weekly x-rays are the most accurate

indication of progress. A stationary or retrogressive course demands re-evaluation of therapy and consideration of surgery.

9. Open drainage may still be applied in the fulminating abscess, the poor risk patient and in the simple uncomplicated acute abscess in which a good result can be anticipated.

10. Resection with antibiotic coverage has reduced the mortality in chronic abscess to a respectably low figure of 1.9 per cent in the reported series of fifty-one cases. Conservation of lung tissue is important, and lobectomy and segmental resection are greatly superior to pneumonectomy. The incidence of empyema in pneumonectomy remains far too high.

11. Careful follow-up of all cases of lung abscess and suppurative pneumonia is imperative. Bronchography is usually indicated. Resection should be considered if appreciable damage persists.

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Air Space Abnormalities of the Lungs (Air Space Disorder)

ISRAEL RAPPAPORT, M.D. AND EDGAR MAYER, M.D., *New York, New York*

Loss of lung structure confined to certain parts of the lungs or involving both lungs diffusely is perhaps best designated by the collective term of air space abnormality. The literature relating to this condition has greatly increased in the past two decades, and from it a number of facts have become apparent: (1) Air space abnormality is a common pulmonary finding and is often seen in patients of all ages from earliest infancy to late senility. (2) It occurs most often as an apparent primary pulmonary disease, i.e., an independent entity. (3) It often develops as a complication of a variety of recognized bronchopulmonary diseases. (4) There is no apparent difference in air space abnormalities occurring with or without associated bronchopulmonary disease. (5) Great confusion prevails in their interpretation as indicated by the increasing terminology which now includes the following: cysts, bullae, cystic emphysema, bullous emphysema, honeycomb lung, vanishing lung, cotton candy lung, pneumoceles, cystic bronchiectases and bronchiolectatic emphysema. (6) In spite of this varied nomenclature there is still obviously a strong tendency to reduce all air space abnormalities to the common denominator of emphysema, and the majority of workers tend to look upon them as manifestations of obstructive emphysema.

In recent publications we discussed some of the problems involved in their interpretation and pointed out the difficulties inherent in their identification with emphysema. We emphasized that current concepts about emphysema are so problematic that to explain air space abnormalities as manifestations of it is just begging the question. Confirming the need to reduce all pulmonary changes observed in them to a common denominator, we have proposed the concept of "air space disorder."

"Air space disorder" is not just another designation, but is instead an explanation of air space abnormalities on the basis of a new concept which arose from our analysis and interpretation of the clinical and pathologic features of a large number of cases, including all forms of the condition in patients of all ages. In this communication it is our purpose to show how the great complexity of air space abnormalities can be simplified and logically fitted into the natural history of "air space disorder."

THE COMPLEX NATURE OF AIR SPACE ABNORMALITIES

Clinical observations have long established the fact that air space abnormalities comprise the following four types of lung changes:

1. *Overdistention* by stretching of air spaces lacking power of elastic recoil: This occurs by a mechanism which requires little explanation. Thoracic traction expands these air spaces in inspiration. Their failure to retract completely in expiration results ultimately in overdistention. However, it is also reasonable to assume that this overdistention must result in proportionate narrowing of the air passages (bronchi) and that this will eventually lead to a point when obstructive mechanisms begin to operate. While the mechanical factors operating in overdistention are quite obvious, their overemphasis has diverted attention from the real problem, i.e., the primary cause of loss of elastic recoil which to begin with makes overdistention possible. This will be discussed later.

2. *Overinflation* of air spaces due to bronchial obstruction: Here we are dealing mostly with a ball-valve mechanism which prevents deflation with expiration. In final analysis too incomplete recoil during expiration occurs here because of air trapping behind the obstructed air passage. Overinflation appears to be a completely mechanical process and under clinical conditions its usual cause is some inflammatory process in the bronchi. However, it may be pointed out here that it is frequently assumed that in overinflation behind

bronchial obstruction, lung structures may become so overstretched as to lose their power of elastic recoil. Overdistention may then replace overinflation even after the bronchial obstruction has ceased to operate. The validity of this assumption will be discussed later.

3. *Atelectasis*, i.e., compression of air spaces by encroachment of adjacent overdistended or overinflated air spaces: Both overdistention and overinflation necessarily lead to atelectasis of adjacent normal air spaces by compression of surrounding lung tissue or by compression or distortion of their air passages. It is particularly true that in overinflation behind obstruction, pressure often exceeds that in surrounding normal air spaces and proceeds at the cost of the latter. It then results in the combination of atelectasis and hyperinflation associated with such lung changes as cysts, bullae, pneumoceles and the like.

4. *Compensatory overexpansion* of non-affected lung areas has been almost completely overlooked, although it is obviously a very important part of the lung changes comprised in air space abnormalities, all the more so since it contributes to the hyperinflation of the lungs as a whole.

Overdistention, overinflation and atelectasis imply loss of function in the affected lung areas. When this becomes significant in extent it must be compensated for by hyperfunction of non-affected lung areas involving their overexpansion.

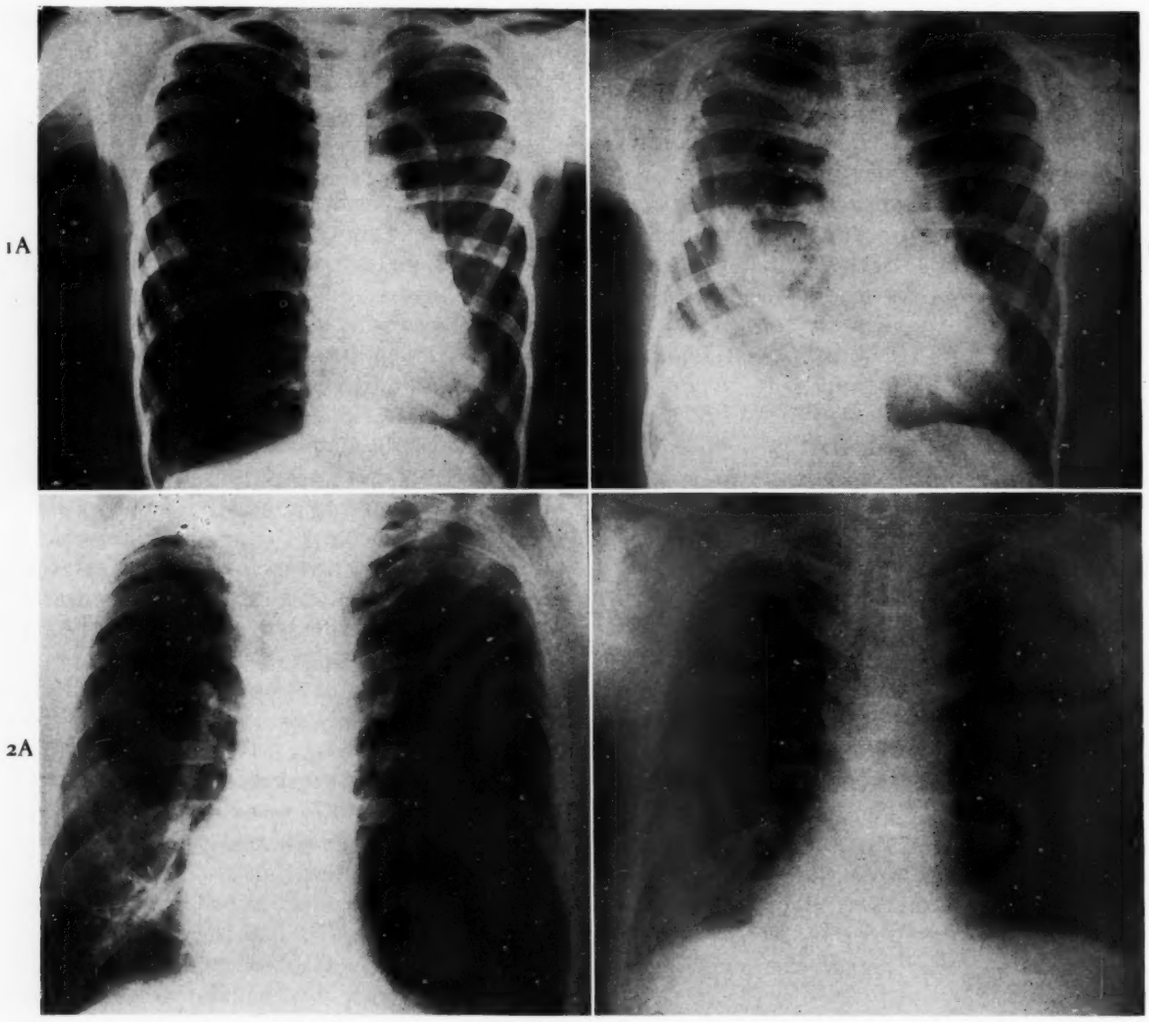
It is a long recognized fact that in the need for increased function the lungs can overexpand for compensatory hyperfunction. This develops temporarily in the "effort" lung and permanently at high altitudes. It has also been amply demonstrated that one lung can compensate for the other; and in the same lung one part likewise can compensate for the other by overexpansion. The latter is a common feature of all pulmonary affections involving sufficiently large parts of one or both lungs to require compensatory hyperfunction of the non-affected parts. Rightly or wrongly this has been designated as compensatory (vicarious) emphysema because of the overexpansion of the hyperfunctioning lung areas. We previously explained this[1,2] by postulating that only a fraction of the air spaces available in any part of the lung are ordinarily expanded for simultaneous function at rest, and all available air spaces will be expanded in compensatory hyperfunction. Compensatory emphysema differs from genuine emphysema in that the latter consists of an increase in the size of the air spaces in the area affected, while the former implies an increase in the number of air spaces of normal size. In the former normal alveolar function is even improved.

Air space abnormalities of limited extent can be compensated for by non-affected lung areas without significant overexpansion of the lungs as a whole and may then be present without clinical symptoms or signs. This explains the clinical latency of air space abnormalities discovered only by chance x-ray or by other accidental changes. Air space abnormalities affecting increasingly larger parts of the lungs must elicit compensatory emphysema in the non-affected parts comparable in extent. Often to begin with the clinical manifestations are due to the extent of the compensatory function of non-affected parts and the significant overexpansion of the lungs as a whole leading to ventilatory disturbance. With continued progress of air space abnormalities, clinical manifestations often arise when the non-affected lung areas are encroached upon so that they are no longer capable of compensatory function even while the lungs as a whole are not overexpanded.

Compensatory emphysema involving large parts of the lungs must be maintained by increased thoracic traction which for obvious reasons aggravates also the mechanical changes in the affected parts of the lungs. This is particularly true in the presence of diffuse air space abnormalities when the advantages gained by compensatory overexpansion are more limited. Therefore it is indeed true that "emphysema begets emphysema." The presence of compensatory emphysema may be difficult to discern in air space abnormalities with overdistention and overinflation of significant extent.

REVERSIBILITY OF AIR SPACE ABNORMALITIES

Among the chief characteristics of air space abnormalities is their variable reversibility. The most complete reversibility is demonstrable in cases of hyperinflation and atelectasis which often occur spontaneously. Caffey[3] has shown that spontaneous reversibility of overinflated lung areas with re-expansion of adjacent atelectatic areas is practically the rule in children. In later life too it often occurs



1A 1B

2A 2B

FIG. 1. A, eleven year old girl. X-ray reveals a pneumocele of enormous size occupying the right hemithorax and even herniating into the left side. It appears as if the right lung had been turned into one vast air sac. B, x-ray taken after operation which involved deflating and resecting a hyperinflated air sac formed in a part of the right lower lobe. The whole right lung was atelectatic due to compression; following operation this re-expanded lung proved to have fairly normal function, even after a decade of atelectasis.

FIG. 2. A, fifty-five year old man known to have bullous emphysema for fifteen years. X-ray reveals an acute pneumocele in the left hemithorax. Required emergency decompression by trocar and continuous suction. B, x-ray shows re-expansion of the left lung which was even better than the status before the onset of the acute pneumocele. At that time the left lung contained extensive air space abnormalities which appeared irreversible.

spontaneously. We have observed this especially in cases in which fluid (probably secretions) accumulated in the abnormal air spaces. As this fluid is eventually absorbed, the air is also absorbed. Reversal can be brought about by surgical deflation, perhaps in a majority of cases. The extent of reversibility of air space abnormalities is quite unpredictable from the clinical and x-ray features. In general it can be said that the more extreme the extent of hyperinflation, the more likely is there extensive mechanical obstructive hyperinflation of a high degree of reversibility. However, the extent of reversibility is often not realized until demonstrated by surgical intervention. (Figs. 1 and 2.)

Reversibility of compensatory emphysema is of course obvious, but its extent will naturally depend on the extent of actual reversal of the changes in the affected lung areas. Clinically the situation is often such that compensatory emphysema must be maintained permanently

and becomes reversible only as the lungs cease to be expanded at death. During life reversibility of compensatory emphysema is responsible for much confusion in the correlation of the clinical and morphologic features in air space abnormalities. Postmortem the extent of air space abnormality is often much less than would be expected from the clinical features, even discounting the expected loss of air from the lung under the circumstances.

The previous brief review of lung changes involved in air space abnormalities should suffice to indicate that their nature is far more complex than would appear from current literature. Not only have the structural changes associated with deflation of adjacent air spaces been neglected, but also what is even more significant, the functional compensatory changes which to a great extent determine the clinicopathologic features have been practically overlooked. The features of air space abnormalities have been misinterpreted chiefly by overemphasizing the role of mechanical factors. Attention has been focused almost exclusively on the structural features associated with mechanical hyperinflation, and these have become identified with emphysema. The result is the current completely mechanical concept of the nature and origin of emphysema.

The error in this interpretation becomes apparent if we consider that, as previously mentioned, the mechanical changes in air space abnormalities are reversible while so-called genuine emphysema is not reversible. As pointed out the mechanical factor involved in overstretching abnormal air spaces operates on structures which have lost their power of elastic recoil. The characteristic feature of genuine emphysema is the primary loss of elastic recoil, and the latter is the crux of the problem of the origin and nature of emphysema.

THE PROBLEM OF THE PRIMARY DEFECT IN AIR SPACE ABNORMALITIES

That current concepts cannot account for the origin of air space abnormalities has been increasingly realized by discerning observers. As was pointed out recently by Head,[4] "The prevailing concept that ball-valve obstruction of a bronchus with consequent air entrapment is the only mechanism which can account for ballooning of intrapulmonary cavities and cysts is incorrect." He contends that "another mechanism has much broader implications in pulmonary physiology. . . . Any intrapulmonary cavity whose walls are stretchable but inelastic, or any portion of lung which has lost its elasticity but not its stretchability, stretches with each inspiratory effort and thus increases gradually in size to the limits of its stretchability and at the expense of the expansion of the normal elastic lung. This principle is less easy to explain or to understand. It is, however, extremely important. It accounts, I believe, for the tremendous increase in the size of congenital pulmonary cysts, in part for the progressive enlargement of blebs and bullae and also for the enlargement of any lobe or segment of a lobe which, as the result of emphysema, has lost its elasticity but not its stretchability."

In most recent publications Churchill,[5] realizing the inadequacy of prevailing concepts, also discussed the problem of "paradoxic development of cystic and bronchiectatic changes following bronchial obstructions." His answer to this is what he describes as the "space-occupying function of air" based on physiologic principles regulating the functional relationship of lung segments. Churchill contends that "despite being deprived of participation in ventilation, the alveoli in diseased segments are found filled with air that enters through adjacent normal segments or remaining normal bronchi within the diseased segment." His explanation of this remarkable phenomenon is particularly significant. "The architectural design of the lung is such that its spatial integrity tends to be preserved even after a morbid insult has destroyed the function of segmental structural units. This mechanism involves what is referred to as the 'space-occupying function' of air within an organism, a physiologic principle illustrated by other examples."

Head too speculates on the important role of collateral ventilation and explains the primary defect as follows: "The air which enters the parenchyma through collateral channels behind obstructed segmental or lobular bronchi does not readily flow in and out but tends to remain in place, thus keeping the parenchyma continually or recurringly dilated until eventually it loses its elasticity and becomes truly emphysematous." Head contends that "this relative entrapment of the air which has entered obstructed lobules and segments,

accounts for the development of emphysema rather than entrapment by ball-valve obstruction of the bronchi, as was predicated by Laennec."

Analyzing these recently proposed views, it seems to us that they are merely adding some new hypothetic features to the mechanistic ideas, and support essentially the old concept of obstructive emphysema as the sole basis of air space abnormalities. Ingenious as these speculations are, they have not solved the problem, namely, the ultimate cause of emphysema and its role in air space abnormalities. However these ideas do point up the difficulties in the current crudely mechanistic concepts and confirm the need to assume the existence of a primary lung defect, on the basis of which abnormal air spaces as well as emphysema develop.

OUR CONCEPT OF THE PRIMARY LUNG DEFECT

Morphologically abnormal air spaces are characterized chiefly by the loss of alveolar parenchymal structures. The air spaces become enlarged by coalescence; they are not distended. In the final analysis air spaces so enlarged represent loss of the normal partitions. If we conceive that the air space partitions are not merely structural fixtures but are in the nature of a functional activity of these structures, then loss of air space partitioning becomes the manifestation of a disturbance in the function of the lungs so conceived. Such a truly biologic concept of "visceral" lung function was presented recently by one of us.[6] Our interpretation of air space disorder as a manifestation of a structural as well as functional lung defect is based on that concept.

This defect may involve certain restricted parts of the lungs or involve both lungs diffusely. Lung areas so affected are not distended, in fact their volume may even be diminished, yet their air spaces are too large in proportion to their fine air passages. Their bronchi are patent and may even be somewhat distended. However, lung areas which have lost their parenchyma and its partitioning function tend eventually to become overstretched by thoracic traction because of loss of power of elastic recoil. Also the bronchus here tends toward oversecretion because of increased arterial blood supply from augmented bronchial circulation. Bronchorrhea invites infection with its sequelae of obstruction. This leads to hyperinflation as a local secondary manifestation of mechanical origin as previously discussed.

THE NATURAL HISTORY OF AIR SPACE DISORDER

As to the origin of the primary pulmonary defect so conceived, the answer, we believe, lies in the natural history of air space disorder as gleaned from clinical observations interpreted in the light of the concepts previously mentioned. We have tabulated what we conceive to be the natural history of air space abnormalities in Figure 3.

As indicated in Figure 3, we postulate that the primary pulmonary defect may be of developmental or acquired origin. The alveolar damage may arise at any time from the very beginning to almost the very end of life. Particularly critical periods are the intranatal establishment of lung function, the postnatal continued growth of the lungs and the decline of functioning capacity with advanced age. In the latter, acquired, and in the two former, developmental defects may be assumed to play the predominant role. In previous publications[7,8] we have discussed reasons which lead us to believe that developmental defects carried into later life play the important role of a predisposing factor even in acquired defect. In turn, acquired pulmonary changes play an important role in promoting the evolution of developmental defects, even in early life, causing these to go on to air space disorder. Thus the pathogenesis of the latter in any phase of life is often best explained on the basis of acquired pulmonary changes, the origin of which can be traced back to persistent developmental defects brought along from very early life.

We have advocated this concept of the long range natural history of chronic pulmonary disease with, or related to, emphysema in several publications in past years.[7,8] The very significant role of developmental defects arising during the postnatal growth of the lungs may now be considered as fairly well demonstrated.[9] Since then much confirmatory evidence has appeared in recent literature.[10]

Recently much evidence has come forward of perhaps even greater significance of the intranatal phase of establishment of lung function at birth. There is now evidence especially incriminating violent resuscitation efforts now practiced with high pressure machines on new-

borns with tardy establishment of lung function.[11]

Lung defects acquired throughout adult life are also quite characteristic of the subsequent phases of life as indicated in Figure 3.

From the foregoing analysis it should be apparent that the great confusion in the interpretation of air space abnormalities resulted from failure of recognizing the roles played by the primary lung defect in the origin, and the role of compensatory emphysema in the evolution of their morphologic and clinical features. Identification of the primary lung defect of developmental or acquired origin with secondary changes of mechanical origin and our inability to separate compensatory emphysema when the latter complicates the combination of the former two prevents better understanding of air space abnormalities.

On the basis of the previous concepts it appears logical to bring all air space abnormalities under one common denominator and we proposed that they be designated by the name of "air space disorder" for the reasons just presented: to wit, we are dealing with a pulmonary disease entity which is both morphologic as well as functional in its origin and its nature. Morphologically it is truly of the nature of a disorder of the air spaces of the lungs comprising a complex combination of distended as well as compressed lung areas. From the standpoint of its functional implications it represents a disturbance in the activity of the structures of the air spaces of the lungs.

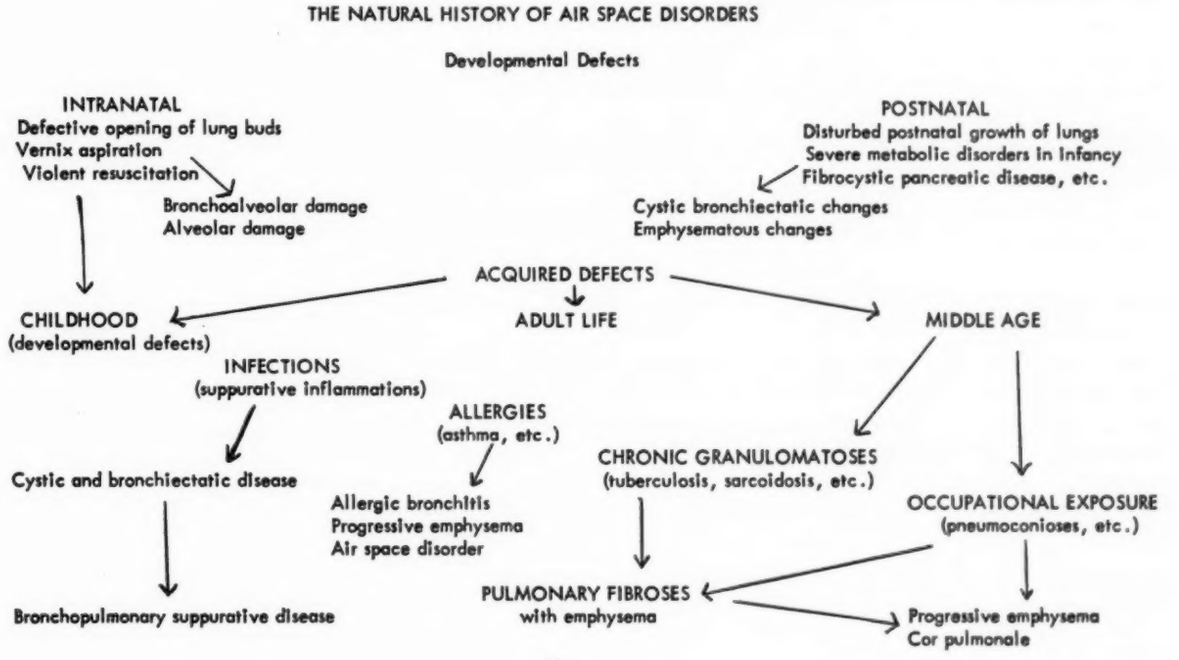


FIG. 3.

PRACTICAL ASPECTS

Understanding of the nature and origin of air space disorder should lead to more frequent recognition and to more correct interpretation of its clinical manifestations. It will account for the occurrence of identical or very similar clinical pictures in a great variety of chronic pulmonary diseases of different etiology. It will explain the phenomena of so-called emphysema arising in any phase of life from earliest infancy to late senility in association with clinical conditions characteristic for these age periods. (Fig. 3.)

Knowledge of the circumstances which lead to development of air space disorder should lead to its earlier recognition by closer observation of progress of postnatal lung development in infants and children. It should lead to deliberate search for lung defects when those should be suspected on clinical grounds or from x-ray features in adolescents and young adults. It should result in correct interpretation of clinical pictures based on air space disorder such as pseudo-asthma and others discussed in previous publications.[7,8] Finally it should

result in earlier recognition of these pulmonary conditions before they end up in vanishing lungs or so-called progressive bullous emphysema of hopeless extent.

Progress in treatment is bound to follow advances in the recognition of air space disorder and in the understanding of its nature. This should begin with progress in prevention of its mechanical complications by such methods as improved technics of resuscitating asphyctic newborns, improved methods of keeping airways open in children afflicted with pulmonary infections, mucoviscoidosis and the like, and resection of localized progressive lung defects when they begin to encroach on adjacent lung areas and interfere with the normal growth and expansion of the latter.

Knowledge of the frequently insidious nature of air space disorder should stimulate progress in assessing the proportion of normal and abnormal lung areas by such methods as angiography, planigraphy, bronchography and bronchospirometry. Correlation of these with ventilatory and gas-exchange studies should yield true estimates of the functioning capacity of the lungs, upon which indications and feasibility of surgical therapy and clinical management in general must be based.

CONCLUSIONS

Interpretation of air space abnormalities has remained confused chiefly for two reasons: (1) Failure to recognize that loss of power of elastic recoil in genuine emphysema must be traced back to a primary lung defect. (2) Failure to consider the role of compensatory emphysema so difficult to discern in presence of genuine emphysema, and of mechanical hyperinflation or overdistention. These have led to identification of air space abnormalities with emphysema and to a concept of the latter in which the structural features and the mechanical factors are overemphasized.

Clinical experience indicates that air space abnormalities are complex conditions, the true nature of which is best described as "air space disorder" with functional as well as morphologic implications.

On the basis of a new concept of "visceral" lung function and its implications regarding the role of developmental postnatal and acquired lung defects, the natural history of all these pulmonary conditions can be readily reduced to the common denominator of "air space disorder."

The practical aspects of this concept in relation to clinical problems are presented.

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Roentgen Manifestations of Pulmonary Tension Disorders in Infants and Children

JOHN A. CAMPBELL, M.D., *Indianapolis, Indiana*

From the Department of Radiology, Indiana University Medical Center, Indianapolis, Indiana.

THE pulmonary tension disorders of infants and children are those characterized by an excessive inflation of the bronchial structure, the lung parenchyma or the pleural space by encysted or trapped air under pressure. The majority of these conditions, particularly the most serious variety, are encountered in the neonatal period or first few months of life. Those occurring later in infancy or early childhood are apt to be less disabling and demand less urgent treatment. The expansile changes frequently, but not invariably, result from some kind of check-valve mechanism between the distended area and its air supply.

All of these tension states are manifest roentgenologically by uniform, distinct areas of intrathoracic radiolucency associated with displacement and compression of adjacent soft tissue structures. Roentgen examination undoubtedly offers the best means for clinical detection and evaluation of the extent of these lesions, yet this method has serious limitations in the differential diagnosis of them. The roentgen findings will not always establish that the tension rarefaction involves the lung parenchyma rather than the pleural space, and frequently will not demonstrate with certainty whether the lesion is a solitary loculated air cyst or has a diffuse lobar emphysematous distribution. Furthermore, it is virtually impossible on roentgenographic grounds alone to distinguish certain naturally reversible acquired cysts from congenital irreversible cysts which need surgical treatment. Nevertheless, serial chest roentgenograms provide a most important study of the progress of the hyperinflated areas which aids in deciding the proper course of management.

Since the natural history of many of these lesions allows them to regress spontaneously while others progressively jeopardize the life of the individual, the need for critical roentgen analysis in every case cannot be overemphasized.

CLINICAL FINDINGS

Clinically, these conditions are usually accompanied by varying degrees of dyspnea and cyanosis. Occasionally, profound respiratory distress due to acute obstructive changes or severe compressive atelectasis of the adjacent normal lung develops rapidly making surgical excision urgent and life-saving. Since such violent and alarming symptoms can be more transient and benign in younger infants than in older patients, the decision concerning the degree of hazard to the infant's life is probably better evaluated by the pediatrician than the surgeon in these instances. Acute respiratory infection may or may not be present as a complicating or secondary factor. More often, however, the infection may constitute the primary cause of the tension phenomenon which may show spontaneous regression after the infection subsides. This is a strong argument for conservative treatment in all infected cases. Spontaneous pneumothorax due to rupture of the hyperventilated area into the pleural space may cause a sudden increase in respiratory embarrassment requiring immediate thoracentesis with an underwater seal.

PATHOLOGIC CLASSIFICATION

Attempts to classify these disorders are largely unsatisfactory in that the basic mechanisms of their production are so poorly understood pathologically. This is partly due to the minute nature of the anatomic structures involved and to the fact that pathologic specimens do not actually reveal functional derangements which may operate in the living organ. The obvious check-valve mechanism which is apparent clinically in many hyperventilated lungs or cystic lesions is very fre-

quently impossible to demonstrate in gross or even microscopic examination of the resected specimens; yet the similarity of appearance and behavior of these conditions to seemingly identical lesions due to an aspirated foreign body in the bronchial tree or a traumatic tension pneumothorax makes the assumption of an underlying check-valve mechanism valid in all cases.

Any classification based on pathologic findings must deal with the controversial problem of whether certain pulmonary cystic lesions are of congenital or acquired origin. Unfortunately, we have not reached a point in our thinking on these entities to say with certainty what constitutes a truly congenital cystic malformation in many instances. Even at surgery, there is no consistent way of differentiating the congenital from the acquired cyst because of the gross similarities and wide variations in the findings. Both types show thick or thin walls, air-fluid content, and epithelial lining. Even the presence of bronchial epithelium, cartilage, mucous glands, elastic and muscle tissue does not conclusively point to congenital origin because it may only represent the wall of a severely dilated bronchial structure distal to an obstruction.

On the other hand, there is little question that such potential causes of tension cystic disease as adenomatoid hyperplasia of lung (cystic hamartomas), cartilaginous hypoplasia of bronchial structures and intralobar sequestration of the lung are of congenital origin.

The real controversy in this matter stems not so much from the disagreement over the pathologic findings as from the fact that the distinction as to whether a lesion is of congenital or acquired origin may be vital to prognosis and treatment. There is a natural tendency to submit to surgical correction disorders attributed to congenital anomaly on the basis that they are irreversible.

On the other hand, surgical excision is more frequently postponed in suspected acquired cystic lesions on the assumption they are more apt to regress spontaneously.

Caffey[1] points out that spontaneous regression without surgical removal is the rule for most pulmonary cysts if their course is followed sufficiently long. Even if the cyst persists after months or years of observation, the patient usually continues to thrive. He believes nonoperable management should be the rule in all pulmonary cysts of infancy except in rather rare instances in which acute respiratory distress due to marked overexpansion of the cyst makes surgical decompression or excision a life-saving necessity.

To avoid needless radical surgery on a tension disorder which may regress spontaneously, operative intervention should be based only upon unsatisfactory clinical progress and never on the roentgen findings alone.

Suffice it to say that from the standpoint of pathologic classification tension disorders of the lung develop through a variety of check-valve mechanisms derived from: (1) developmental bronchopulmonary malformations; (2) bronchogenic pulmonary suppuration; (3) intrabronchial and extrabronchial occlusions; and (4) pleural fistulas.

ROENTGENOLOGIC FINDINGS

Accurate roentgenologic differentiation of these various pathologic groupings is frequently impossible because of the similarity in findings. Nevertheless, identification is considerably enhanced by carefully correlating the roentgen appearances of the lesion and its anatomic distribution with the factors of age, mode of onset, infection and collateral disease processes. Anatomically the involvement may be unilateral or bilateral in distribution. The expanded area may be intralobar, lobar or pneumonic in extent with those showing a lobar emphysematous character tending to be more serious and irreversible. The age and history of onset helps to evaluate the origin of the process, i.e., occurrence at birth, trauma, foreign body aspiration. The association of current or previous inflammatory disease makes some roentgen evidence highly specific. A knowledge of coincidental cardiovascular, mediastinal, systemic, or other organ disease process may give the roentgen findings greater specificity. Roentgen diagnosis should not be difficult in a great many instances if one is aware of the entity and its attendant manifestations.

1. *Developmental Bronchopulmonary Malformations.* Probably the most frequent lesions in this group are the lobular bronchial cysts. They seem to result from embryonic blockage in the lumina of the bronchial buds, and develop as single or multiple cystic cavities lined with bronchial structures and filled with secretions. Norris and Tyson[2] have shown by an ingenious study of serial sections that the

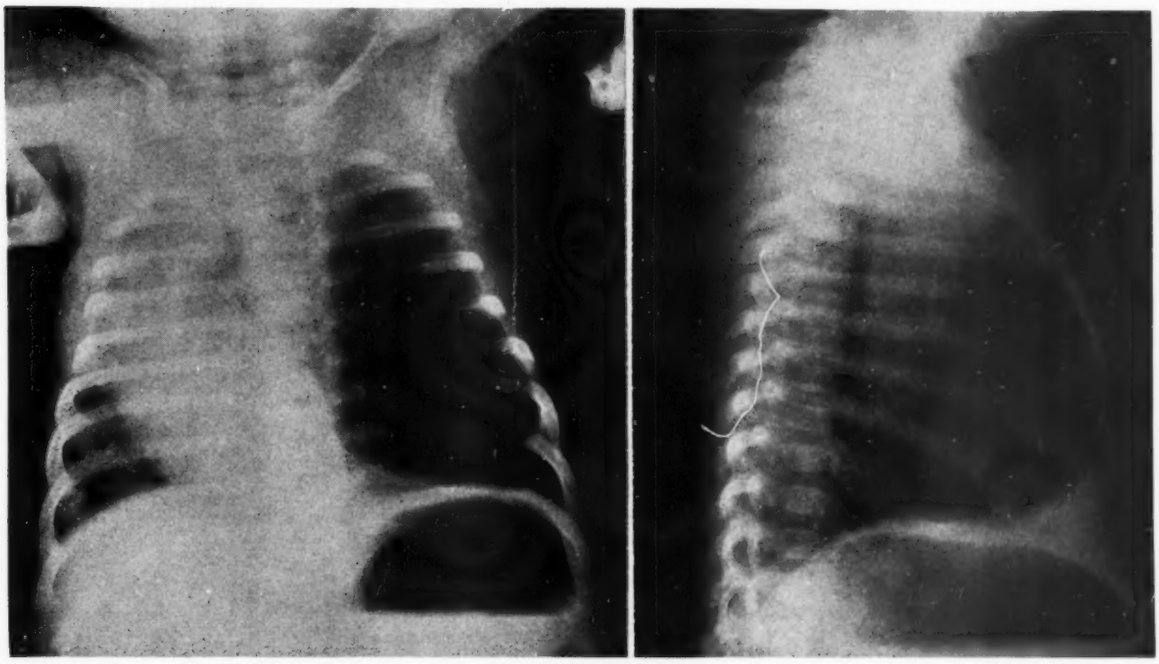

FIG. 1. Solitary bronchial cyst. A spherical, trabeculated tension cavity expands the left lower lobe. Its thin wall is barely discernible. Note compressed lung segments in the angles of the thorax. This proved to be a loculated bronchiectatic malformation.

original defect is a small irregular dilatation in the smaller bronchioles. When arrested in this stage, such focal dilatation results in fetal or cystic bronchiectasis characterized by rather uniform-sized, thin-walled cysts in the terminal arborization of the bronchial tree.

If the process continues, these bronchiolar dilatations become pinched off to form larger cysts, up to several centimeters in diameter. When diffuse, this process gives rise to the so-called honeycomb lung and may involve one or several lobes. This type of honeycombed lung is actually a form of bronchiectasis and is clearly demonstrable by bronchography in contradistinction to the acquired fibrocystic form of honeycombed lung in which the check-valve alveolar and interstitial cysts do not fill on bronchography.

Most frequently, the focal segmentation process is isolated and a single bronchial cyst forms. The postnatal behavior of such a cyst depends on mechanical factors. When a check-valve mechanism develops in the narrow tortuous bronchial lumen, the fluid is evacuated and the cyst may expand rapidly to form a huge tension cavity which greatly distorts the lobe in which it arises. Roentgenographically, such a solitary bronchiectatic tension cyst shows a clear or trabeculated large area of rarefaction, with discernible lung tissue at the apex and angles of the thorax, and absence of collapsed lung at the hilum. Mediastinal herniation, when present, is much less striking than with tension pneumothorax. The cyst will usually not visualize on bronchography. The margin of the cyst wall is generally not clearly distinguished unless it is secondarily infected or contains fluid. Anatomically, such lesions are intralobar in extent. (Fig. 1.)

A less frequent type of bronchopulmonary malformation is cystic hamartoma or adenomatid hyperplasia of the lung[3] observed almost exclusively in premature infants. It is characterized by a marked proliferation of embryonic lung tissue resembling normal adult lung tissue except that it is composed of an organoid overgrowth of terminal bronchioles surrounded by many tiny alveolar lobules. The malformed portion passes directly into normal-appearing lung tissue at the periphery without any demarcation or encapsulation. The essential feature is an excessive overgrowth of bronchioles, which causes lobar enlargement, while the development of the alveoli is completely suppressed except at the periphery.

This mass may become hyperventilated under tension, or remain completely distended with secretions, depending on the potency of

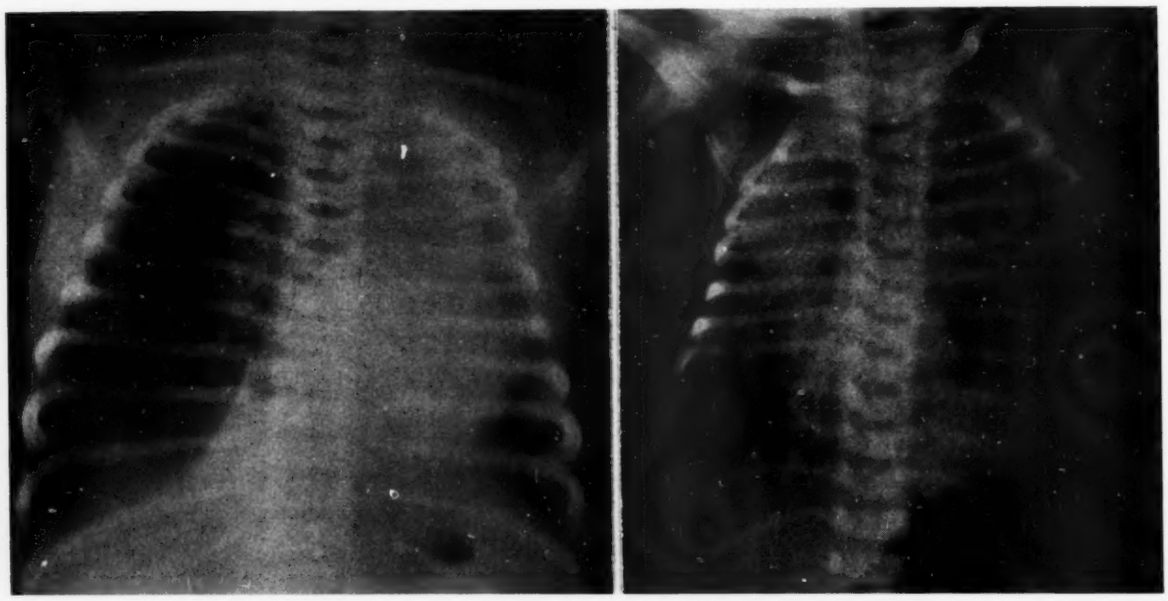

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FIG. 2. Cystic harmartomas. A, communicating type of adenomatoid hyperplasia of right lung in which a check-valve hyperventilation of the involved right upper lobe has occurrred. B, non-communicating type of adenomatous hyperplasia of lung showing a dense cystic mass expanding the left upper lobe and filled with fluid secretions instead of air because no bronchial communication has been established.

its bronchial communication. A check-valve mechanism in the faulty and undersized bronchial supply causes a roentgen picture that is often indistinguishable from obstructive lobar emphysema caused by bronchial occlusions. Its occurrence in the premature newborn without evidence of infection or aspirational episode together with the presence of hamartomatous mass at the hilum should suggest the diagnosis. Usually this type of lesion is not capable of producing the extreme degree of expansile distortion of the hemithorax that is seen with the locular cysts. There is also no formation of large air cysts because the check-valve mechanism occurs in the larger bronchi. (Fig. 2.)

Another type of bronchial malformation which may show tension manifestations is sequestration of the lung.[4] The pulmonary abnormality consists of lung tissue which is sequestrated or dislocated from the normal bronchial and blood supply of the surrounding lung. This occurs as either an intralobar or extralobar sequestration depending upon whether the anomalous segment lies within the lower lobe or adjacent to it forming an accessory lobe or lung. The bronchial supply of the abnormal lung does not communicate with that of the involved lobe, and its blood supply is derived from an aberrant pulmonary artery arising directly from the thoracic or abdominal aorta. This artery may pass through the diaphragm or mediastinum to reach the lung creating an important hazard if lobectomy is contemplated. In intralobar sequestration the venous drainage is to the normal pulmonary veins, and in extralobar sequestration to the systemic veins.

The sequestrated lung may consist of one or more large bronchogenic cysts or a polycystic mass showing expansile characteristics incident to infection or bronchial occlusion. Radiographically, this lesion may remain obscure until complicated by infection when pneumonic density surrounding air and fluid distended cysts develops in the posterior, medial, inferior portion usually in the lower lobes. Sometimes a finger-like extension of the density passes medially toward the diaphragm and aorta. Although the tension characteristics of the cystic disease show spontaneous regression as infection subsides, the tendency for progressive expansion with repeated inflammatory episodes may make surgical removal desirable. Bronchography and barium swallow aid to differentiate this condition from suppurative bronchiectasis and diaphragmatic hernia. Aortography shows the associated vascular anomaly.

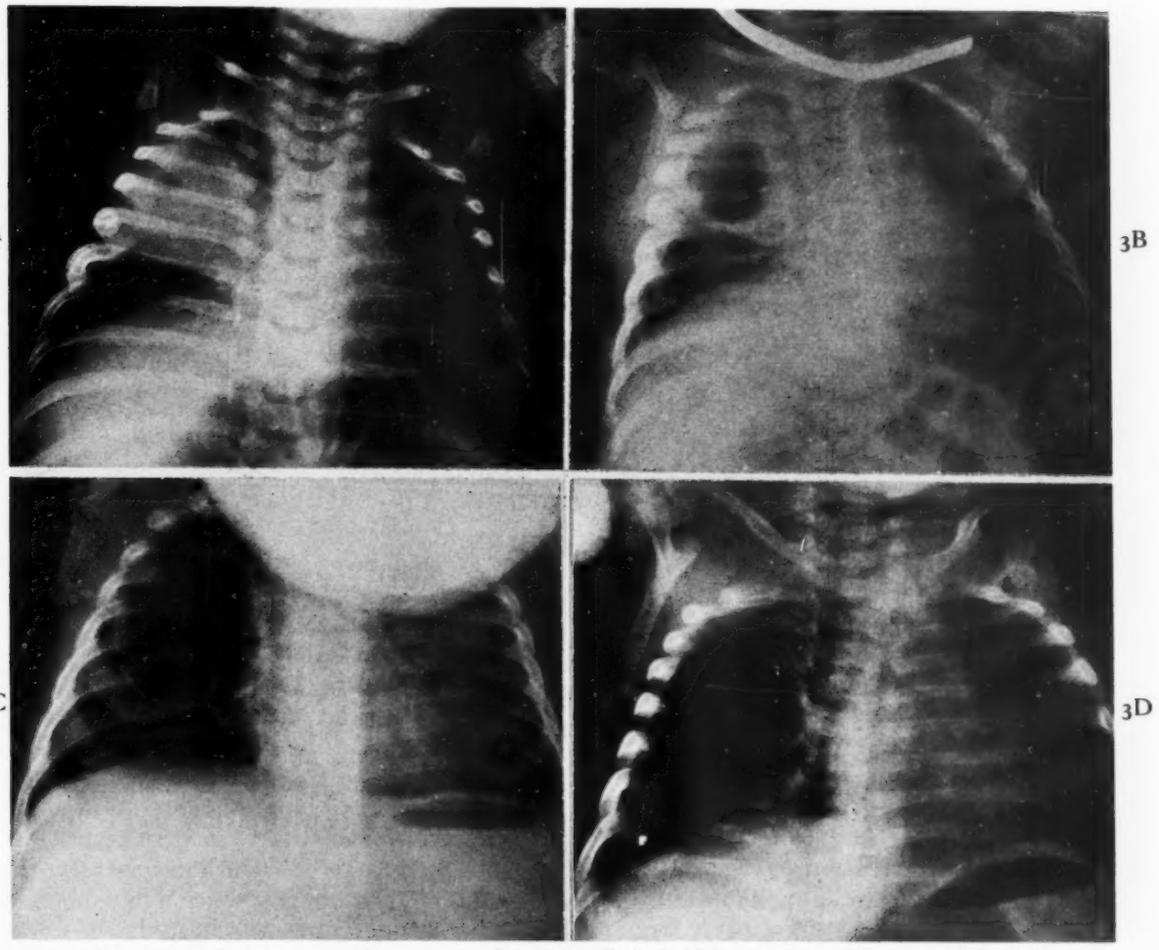

FIG. 3. Pneumatocele in suppurative pneumonia. A, small air bubble appears within consolidated lobe. B, definite cavernous area appears one week later surrounded by wide band of density. C, two weeks later the tension cyst has expanded markedly following resolution of the pneumonic infiltrate. D, at four weeks the pneumatocele is further distended and produces mediastinal displacement. The lungs show compensatory emphysema.

2. *Bronchogenic Pulmonary Suppuration.* Suppurative bronchopulmonary disease is the chief cause of acquired intralobar tension cystic disease. During the course of the bronchogenic spread of certain forms of interstitial pneumonia, one or more round hyperventilated cavities or pneumatoceles may appear roentgenographically within the infected portions of the lung parenchyma. These may rapidly enlarge and remain as thin-walled, air-containing cysts for many months after the pneumonic density has disappeared. Doub,[5] Caffey[6] and others believe that these radiolucent areas constitute a special form of regional obstructive emphysema resulting from a check-valve mechanism produced by thick exudate, edema and expiratory constriction in a small bronchus supplying an area of interstitial pulmonary sepsis and necrosis. As the necrotic tissue is liquefied and evacuated through the bronchus, the resultant cavity is increasingly expanded with air. These cavities may contain fluid levels and be mistaken for necrotic abscesses. They are lined with a thin layer of granulation tissue.

Tension pneumatoceles occur most frequently in staphylococcic and other suppurative pneumonias,[7] but they are also seen in tuberculosis (caseating bronchopneumonia) and similar granulomatous infections. In fact, whenever a pneumatocele of this type is encountered, staphylococcic pneumonia should be strongly suspected as the cause. They tend to disappear spontaneously as the infection subsides, although they frequently persist for several months after disappearance of the

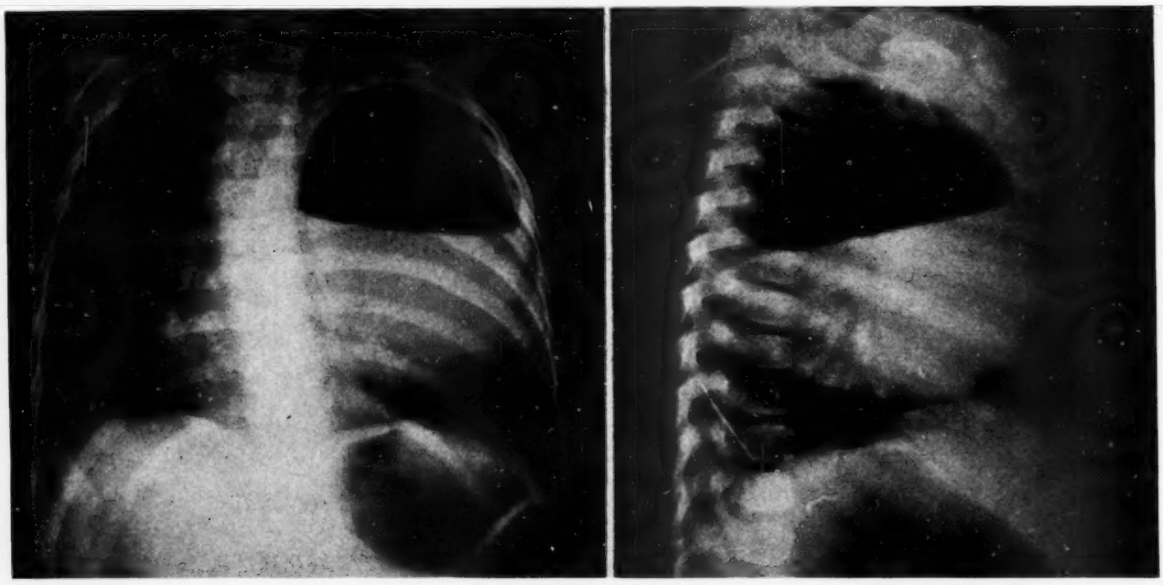

FIG. 4. Tuberculous tension pneumatocele. This huge high tension air-filled cavity containing fluid proved to be a regional obstructive emphysematous cyst in the left upper lobe lined with tuberculous granulation tissue. Such lesions occur in caseating bronchopulmonary tuberculosis and may undergo spontaneous regression during the course of disease.

inflammatory densities. Only at this stage is a problem in differentiation created, since there are usually no roentgen clues of previous inflammatory disease. Following collapse or gradual reduction in the size of the cyst, the walls ultimately become impossible to find radiographically. It is not uncommon for one cyst-like area to disappear while one or more similar areas of rarefaction develop. This indicates the different stages of pneumonic involvement incident to bronchogenic spread of these infections.[8]

Roentgenologically, the pneumatocele may be distinguished by the rather characteristic changes in its appearance on a sequence of films. In the initial consolidative phase of the pneumonia, such a lesion resembles air bubbles deeply seated in the parenchyma. During the phase of consolidation a small poorly outlined spherical cavity is seen. As the consolidation resolves, a vivid thin-walled pneumatocele is formed that is at first surrounded by a wide margin of lung tissue, but which may rapidly inflate under tension to fill the entire lobe or hemithorax. With this, there may be extreme compression of the adjacent lung and mediastinum. The pneumatocele may vary greatly in size from day to day, occasionally ballooning up to alarming proportions. Suddenly it may disappear entirely because of loss of the valvular mechanism in the bronchus with immediate re-expansion of the surrounding lung. (Fig. 3.)

Only if the tension pneumatocele reaches such proportions that it persistently and seriously embarrasses cardiorespiratory function, or ruptures to form tension pneumothorax is surgical intervention indicated. Here closed catheter drainage will usually suffice, and recourse to lobectomy is rarely necessary. (Fig. 4.)

Intrabronchial and Extrabronchial Occlusions. The classic form of obstructive emphysema of infancy is derived from a check-valve impaction of an aspirated non-radiopaque foreign body, frequently a peanut, into a secondary bronchus. The hyperventilation may be lobar or multilobar in extent and causes displacement of the mediastinum to the side opposite the lesion on radiographs made in the expiratory phase of respiration. The tension state lasts for forty-eight to ninety-six hours and is followed by atelectasis of the involved area. History is important but not always accurate. Age is a factor in that such aspiration generally occurs in a child who has incisor teeth, but no opposing molars, usually about the second and third years of life. This point aids in differentiating the foreign body type from those lobar obstructive emphysemas occurring in the first few months of life. The latter types show a progres-

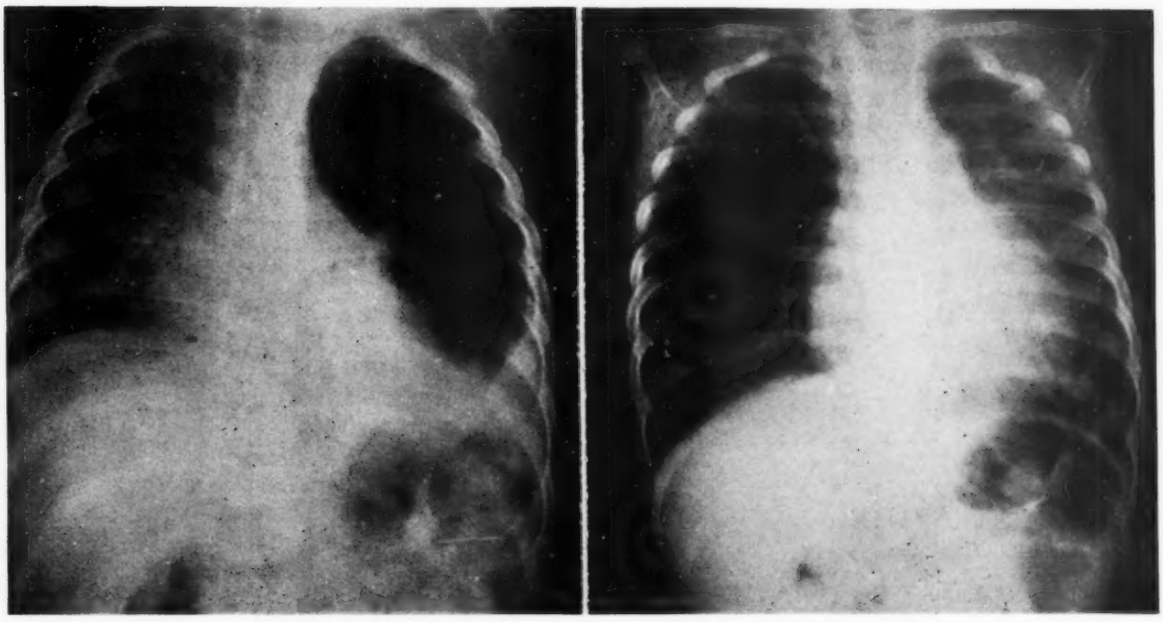

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FIG. 5. Lobar obstructive emphysemas. A, lobar emphysema of left upper lobe due to intrabronchial occlusion incident to hypoplasia of bronchial cartilage. B, lobar emphysema of right middle and lower lobes due to extrabronchial occlusion produced by vascular compression in Eisenmenger's complex. There is atelectasis of the right upper lobe.

sive pneumatic distention of one lobe to extreme proportions causing marked respiratory distress and cyanosis without subsequent atelectasis of the involved lobe. This is probably because the intrabronchial edema at the point of obstruction is less severe than with foreign body. There is also a tendency for involvement of the upper lobes in contradistinction to the lower lobe involvement with foreign body aspiration.

Severe lobar emphysema of this type[9] probably results most frequently from hypoplastic development of the bronchial cartilaginous rings. It is not unusual, as reported by Van Epps and Davies,[10] for the surgeon and pathologist to find no definite obstructive mechanism to explain the marked inflation of the lobe. The overdistended lung shows fragmentation of the interstitial elastic tissue and ruptured alveolar walls from the increased intra-alveolar tension, but nothing to suggest adenomatoid hyperplasia. It has been postulated that an infolding of bronchial mucosa or kinking of an underdeveloped bronchial ring might serve to produce functional check-valving in these instances. Compression atelectasis of the adjacent lobes may be seen in the changing roentgen picture. Spontaneous regression of the tension state, if it does occur, is transient, and fulminating recurrences are the rule. Conservative decompressive measures by needle or trocar have been unsuccessful.

Extrabronchial compressive occlusions cause severe degrees of obstructive emphysema in a single lobe or the entire lung depending upon whether they affect a primary or secondary bronchus. The common causes in infancy are intrathoracic vascular anomalies and mediastinal mass lesions lying close to the bronchus. Duplication and bronchogenic cysts attached or incorporated into the bronchial wall may give rise to a ball-valve action which prevents normal expiratory function and builds up a high tension form of emphysema. (Fig. 5.)

In infants with congenital cardiovascular disease, a persistent ductus arteriosus may overlay the left main bronchus and produce the check-valve effect. Striking examples of extrabronchial interference with the ventilation of the lung have also been seen in cases of aberrant pulmonary veins, truncus arteriosus, vascular ring anomalies, Eisenmenger's complex, transposition of great vessels, and enlarged bronchial arteries.

In these cases one must be careful not to mistake profound avascularity of the lung for

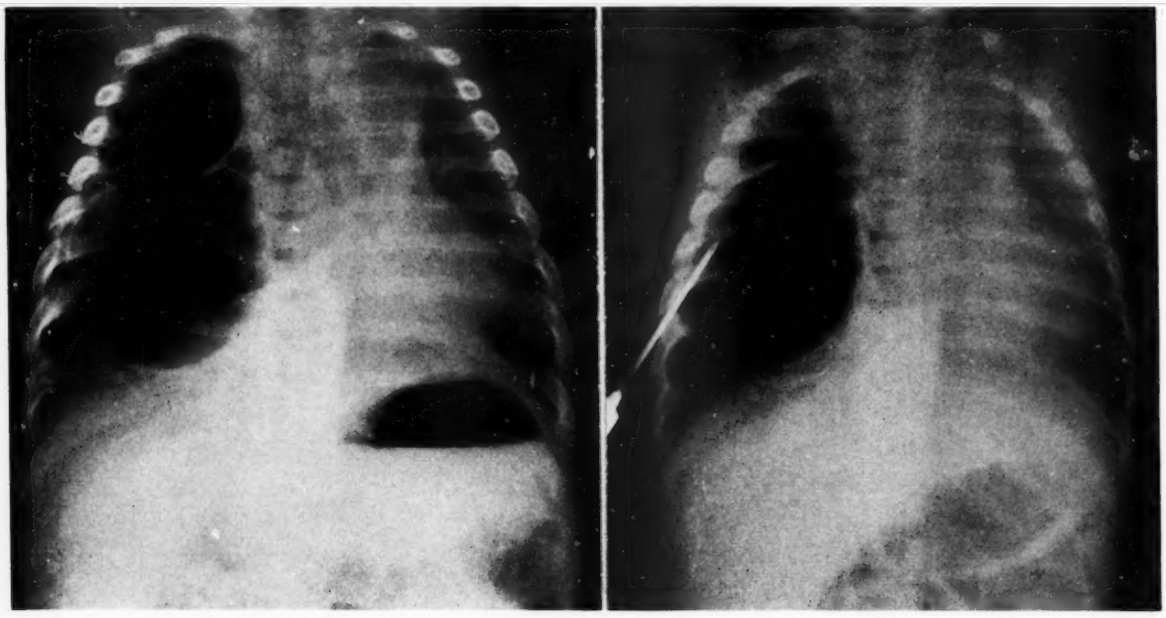

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FIG. 6. Tension pneumothorax and pneumatocele. A, multiple large cystic cavities showing extreme tension phenomena in an infant with suppurative pneumonia. B, following decompression of the larger cavity which proved to be a tension pneumothorax, several hyperinflated parenchymal air cysts remain to cause continued respiratory distress.

emphysema radiographically and vice versa. In emphysema the pulmonary arterial channels are extremely small in appearance due to the high intra-alveolar tension, but the lung volume is increased. When there is primary deficiency in the pulmonary arterial supply, the lung volume is diminished. Paradoxically, the pulmonary arterial shadows may actually appear smaller roentgenologically in severe obstructive emphysema than in pulmonary stenosis. Of course, some extrabronchial occlusive lesions compress the pulmonary artery and the bronchus producing a lung picture almost devoid of vascular markings.

The tension disorders produced in this group may be the most severe of all, and patients are often in such precarious clinical condition as to require emergency surgery. Recourse to bronchography and angiocardiography is frequently not possible, although these measures are obviously useful in differential diagnosis. On plain film studies one may frequently differentiate these diffuse obstructive emphysematous types from the clear rarefactions produced by intralobar pneumatoceles, and solitary locular cysts by the poorly defined margins and lack of definite cavity formation. There is no free pleural margin to the collapsed adjacent lobes as in tension pneumothorax, nor as striking a degree of mediastinal herniation. Obstructive lobar emphysema is practically impossible to distinguish radiographically from adenomatoid hyperplasia and the larger cystic bronchial malformations.

Pleural Fistulas. The non-traumatic tension pneumothorax forms when a check-valve bronchopleural fistula develops incident to rupture of a suppurative parenchymal process or tension cystic lesion in the periphery of the lung. Although such a pleuropulmonary perforation may occur in distended bronchial cysts, subpleural blebs, fibrocystic disease and obstructive emphysema, by far the majority of cases found in infancy develop from an underlying suppurative lung disease.[11] The process is attributed to rupture of a tiny subpleural abscess or pneumatocele in check-valve communication with a smaller bronchus. The development of empyema is probably the first evidence of this type of pleural fistula, and small degrees of pneumothorax may be found roentgenologically in the course of empyema formation if searched for on expiratory films. Sudden tension pneumothorax may occur with

explosive violence causing the infant to suffer a profound and alarming increase in respiratory difficulty. Roentgen detection of such a tension pyopneumothorax in an infant approaches pathognomonic significance in the diagnosis of suppurative pneumonia.[12]

The suppurative lesions are chiefly due to Staphylococcus aureus, but other bacterial, fungous and even parasitic organisms (hydatid)[13] capable of pulmonary sepsis and necrosis may produce identical changes.

Roentgenologically, tension pneumothorax fills the hemithorax with an extremely clear area of radiolucency which flattens the collapsed lung against the mediastinal and produces a sharply defined herniation of the pleural sac across the anterior mediastinal space. When adhesive pleuritis has occurred, the tension pneumothorax cavity may be partitioned into cyst-like compartments by the pleural adhesions and mistaken for large intrapulmonary emphysematous cavities.

Frequently, in an infant with extreme respiratory embarrassment due to staphylococcic pneumonia and evidence of multiple high tension air-filled cystic cavities, the differentiation between multiple distended pneumatoceles and loculated tension pneumothorax is impossible roentgenologically. The differentiation may be aided by direct needle aspiration of the cystic cavity. In tension pneumothorax the lung re-expands and the cavity disappears; while if a pneumatocele is aspirated, the other distended cavities will expand to refill the space. To further complicate this distinction, however, it is possible to encounter a severe tension pneumothorax superimposed on markedly expanded interstitial air cysts. This combination together with the diffuse emphysema in the remaining lung tissue caused by the bronchogenic infection causes a most critical situation for the patient often necessitating emergency surgical decompression. (Fig. 6.)

Besides the simultaneous occurrence of cavernous tension lesions in the lungs and pleural space is the possibility of pulmonary air cyst associated with pleural exudate but without air in the pleural space. Here again, two mechanisms are operating to produce disability from excess intrathoracic tension.

Pleuropulmonary fistulas of purely traumatic origin are relatively rare in infants and easily diagnosed as a rule.

SUMMARY AND CONCLUSIONS

The primary causes of pulmonary tension disorders in infants and children result from check-valve mechanisms, derived from developmental bronchopulmonary malformations, bronchogenic pulmonary suppuration, intrabronchial and extrabronchial occlusions, and pleuropulmonary perforations. Many of these conditions are spontaneously reversible regardless of their derivations, and a conservative non-operative approach should be followed until the irreversible nature is firmly established or critical respiratory distress makes surgical intervention urgent.

Serial chest roentgenologic studies provide the best means of detecting these lesions and evaluating the proper course of management. Although accurate roentgen differentiation of these tension disorders is frequently impossible because of the similarity of findings, the roentgen manifestations in many instances will be sufficiently decisive to allow satisfactory identification of the process. Careful correlation of the roentgen appearance of the lesion and its anatomic extent with factors of infection, collateral disease and age coupled with an awareness of the pathologic possibilities greatly improves the specificity of diagnosis.

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T. C. Case believes that we should not routinely advise radical mastectomy for breast cancer. The average case with metastases or lymph node involvement is frequently inoperable and radical surgery is followed by a high percentage of recurrences. Even when no enlarged glands are palpable, the condition may be inoperable because of involvement of lymphatics in the chest cavity. When the disease is strictly limited to the breast, a radical operation is unnecessary and if performed, may be followed by lymphedema of the arm, causing considerable disability. If the growth is radiosensitive, radiotherapy alone or combined with simple mastectomy suffices. I have had several such cases with good results. (*Richard A. Leonardo, M.D.*)

Cystic Disease of the Lung

WITH EMPHASIS ON EMPHYSEMATOUS BLEBS AND BULLAE

JEROME R. HEAD, M.D., *Chicago, Illinois*

PULMONARY cysts, emphysematous blebs, emphysematous bullae and extreme emphysema localized to a lobe or a segment of a lobe are distinct pathologic entities which have similar clinical and roentgenographic manifestations. All are air-containing cavities within the lung or beneath the visceral pleura. All tend to increase in size, either rapidly or gradually, and all may become so large that they crowd and compress the adjacent normal lung and in this manner produce dyspnea. All are amenable to surgical treatment. The pathogenesis of all except the cysts is poorly understood, and why all of them, even the cysts, grow steadily larger is not generally recognized.

DEFINITIONS AND GENERAL CONSIDERATIONS

Pulmonary cysts are congenital epithelium-lined cavities. There are three varieties, i.e., simple cysts, batrachian cysts and bronchiectatic cysts or congenital cystic lung. Simple cysts are solitary epithelium-lined cavities which have no well organized communications with the air passages. They are often filled with fluid or, if a communication with the air passages has developed, with air or fluid and air. They do not tend to increase in size. They are relatively innocuous, becoming clinically important only when they produce hemoptysis or when they become infected.

Batrachian cysts are segments of lobes whose development has become arrested in the batrachian stage. A patent segmental bronchus enters them and then branches to spread, medusa-like, over their lining surface. Along each such bronchial radicle are numerous openings for the ingress and egress of air. These cysts tend to increase rapidly in size, probably soon after birth. One that is small at birth may in a few weeks come to fill an entire hemithorax, compressing the adjacent normal lung and forcing the mediastinum far to the opposite side.

Congenital cystic lung closely resembles bronchiectasis. It is the result of arrest of development in a late stage. The larger bronchi branch normally but instead of decreasing steadily in size at some point along their course open up into smaller or larger bronchiectatic cysts. From a clinical point of view the disease is scarcely distinguishable from bronchiectasis. The symptoms and treatment of the two conditions are the same. They will not be considered in this paper.

Emphysematous blebs are air-filled blisters covered by the visceral pleura. They are not usually associated with vesicular emphysema. They are produced by the rupture of alveoli into the subpleural plane. Small ones, such as cause most cases of spontaneous pneumothorax, are common. Some of them increase progressively in size. Giant blebs (either single or multiple, either apical or basal) which cause important dyspnea are relatively common. When one observes the interior of these blebs through a thoracoscope, the mechanism which causes them to grow progressively larger is obvious. On inspiration multiple small stomas in the lung open to permit the entrance of air. On expiration they close to prevent its exit. It is obvious that this ball valve mechanism entraps air in them under pressures which on exertion or coughing must be extremely high.

Emphysematous blebs rarely become infected. The author has seen one case of empyema of bilateral blebs. Treatment by rib resection and drainage resulted in the re-expansion of the lung and the obliteration of the blebs. Occasionally serous fluid forms in them. When it does, the blebs usually disappear as the fluid is absorbed. It is probable that the inflammatory exudate closes the communications with the air passages. More will be said about this in the discussion of the treatment.

Recently Korol has presented statistics which indicate that blebs predispose to bronchiogenic carcinoma. The author has seen several cases.

Extreme lobar emphysema in infants and small children is an obscure and dramatic condition in which a single lobe which is grossly normal becomes so tremendously dilated that it occupies an entire hemithorax, compressing the adjacent lobes and pushing the mediastinum far to the opposite side. In recent years an increasing number of such cases has been reported. A number of these patients have been operated upon and a relatively normal respiratory status restored by resection of the emphysematous lobe. The etiology and mechanism of the condition is obscure. It is generally believed that the dilation of the lobe is caused by a ball valve obstruction of the lobar bronchus. The confusing thing is that in only one of the cases has an obstruction been found, either by bronchoscopic examination or by examination of the lobes removed at operation or autopsy. In one case Holinger found strands of tissue partially obstructing the bronchus. Removal of these through the bronchoscope corrected the condition. In no other case has any obstruction been demonstrated. Folds of mucosa and abnormalities of the cartilages have been suggested as accounting for obstruction, but these explanations are hypothetic and probably incorrect. It is possible that bronchial obstruction is not the cause. In the ordinary case of locked emphysema such great dilation of a lobe or lung does not and cannot occur. Usually the greatly narrowed bronchus widens on inspiration to permit the entrance of air and closes on expiration to prevent its egress. For this reason the lobe or lung enlarges only to its inspiratory maximum and it is because it does so that the roentgenogram taken on deep inspiration fails to disclose the condition. Any difference in the aeration of the lungs becomes apparent only on a film taken on expiration. Such great dilation of a lobe as is found in this condition presupposes an obstruction which permits air to enter only when the intrabronchial pressure is greatly elevated as in coughing or straining, but which is complete during normal inspiration and expiration. One can conceive of such an obstruction and can conceive that it might produce a locked emphysema of high pressure. If such an obstruction does ever exist, one would expect to encounter it in other conditions and in adults. Therefore, inasmuch as the very existence of this type of ball valve is problematic and no obstruction of any kind is found in these cases, one is justified in suspecting that some other mechanism accounts for these greatly dilated lobes. The theory that a congenital or acquired loss of elasticity permits them to stretch to this size will be developed in a separate section of this paper.

Emphysematous bullae are air sacs within the lung parenchyma which are produced by the breaking down of the interalveolar septa. They are an end result of severe vesicular emphysema. They are often confused with blebs, and the terms "bleb" and "bulla" are often used loosely and interchangeably. The conditions are distinct, both in their etiology and pathogenesis, and their treatment is entirely different.

Vesicular emphysema is a dilation of the alveoli. It is either caused by or always results in a loss of pulmonary elasticity. Its etiology and pathogenesis are obscure. It is discussed here as a disease amenable to surgical treatment because it is often localized to lobes or segments of lobes. These, because of the entrapment of air and because their loss of elasticity permits them to stretch to such size that they crowd and compress the adjacent normal or more nearly normal lung, frequently act to produce dyspnea in the same manner as do giant cysts and blebs. Not only are such emphysematous lung portions functionless in the exchange of gases, but also by occupying space and taking their share of the pulmonary circulation they limit the maximum function of the more normal lung portions. Resection of such lobes or segments is occasionally indicated and in carefully selected cases may result in an important increase in functional vital capacity.

CONSIDERATIONS OF THE ETIOLOGY OF EMPHYSEMA AND THE CAUSES FOR PROGRESSIVE ENLARGEMENT OF CYSTS, BLEBS, BULLAE AND EMPHYSEMATOUS LOBES

I have already stated that the etiology and pathogenesis of vesicular emphysema are obscure. Pathologically, as first noted by Laennec, the lungs are dilated, they are inelastic and do not collapse when removed from the thorax. The alveoli are enlarged and interalveolar septa are here and there broken down to form smaller or larger bullae. Laennec suggested that the condition was produced by ball valve obstruction of the bronchioles by viscid secretions. He noted, however, that occasionally cough and secretions were absent.

The modern conception of the etiology and pathogenesis of vesicular and bullous emphysema is similar to that advanced by Laennec. It is spoken of as obstructive emphysema, and obstruction of the finer air passages either by secretions, edema or spasm is predicated. There is considerable evidence to support this theory, but that the process is as simple as this seems improbable. It is true that most patients afflicted with this condition have had a chronic cough for many years prior to the development of the emphysema. Without having definite asthma, most of them have a prolonged expiratory phase, some wheezing and a reduction in maximum breathing capacity which is out of proportion to the reduction in vital capacity. These all speak for expiratory obstruction of the finer air passages, as does the fact that antispasmodic drugs increase the vital capacity. On the other hand, some patients have no cough or expectoration, and in bronchial asthma, the condition which would seem most likely to produce it, severe emphysema is rare. Most patients do not have and have never had bronchial asthma. It is also difficult to understand how a ball valve obstruction which permits air to enter the alveoli more easily than it can leave them would result in such extreme dilation. In locked emphysema of this type the alveoli can be dilated only to the inspiratory maximum. It seems probable that dilation beyond this point necessitates a mechanism whereby air is forced into them during forced expiratory efforts. There is reason to suspect that the collateral channels between segments and lobules are routes by which air can thus be forced into obstructed lobules and segments. It is generally believed that when a segmental or lobular bronchus is obstructed, the lung distal to the obstruction becomes atelectatic. This is not true. What is usually interpreted as segmental or lobular atelectasis is merely infection secondary to the interference with the normal drainage of secretions. If the obstruction becomes complete before infection develops, the segment continues to receive air from adjacent segments and probably becomes emphysematous rather than atelectatic. It seems probable that air can flow through these intersegmental communications on cough and other strong expiratory efforts, and that it is in this manner that obstructed segments and lobules become dilated beyond the maximum inspiratory limit. We have observed through the thoracoscope inserted into blebs that the small air passages at the periphery of the lung do open during expiration and close during inspiration. This suggests that the same paradoxic mechanism may operate between lobules. In this connection it seems worth while to report two instances in which complete obstruction of a segmental bronchus resulted in severe emphysema of the lung distal to the obstruction. In one instance we operated upon a young man for what appeared to be a large bleb or cyst at the base of the right lung. This had forced the diaphragm down and had crowded the uninvolved normal lung upward. At operation it was found that there was a tremendously emphysematous accessory lobe which hung from the lower medial margin of the lower lobe by a pedicle of normal lung tissue. The bronchus which should have led into the normal bronchial tree of the accessory lobe ended blindly in the pedicle and the bronchi of the accessory lobe were filled with a pinkish, gelatinous material. The accessory lobe was, however, greatly dilated; and since the only air communications between it and the lower lobe were in the parenchyma of the pedicle, one must suppose that air entered through these on strong expiratory efforts more readily than it could leave through them on normal expiration.

In the second case the bronchogram revealed complete stenosis of the lower lobe and lingular bronchi. At operation the lower lobe was, as would be expected, completely atelectatic. The lingula, however, was markedly emphysematous. That this was more than a mere compensatory enlargement of the lingula secondary to atelectasis of the lower lobe was evidenced by the fact that the dilated lingula had actively crowded the remainder of the upper lobe into the dome of the pleural cavity.

These two cases seem to demonstrate that when in the absence of infection a segmental or lobular bronchus is obstructed and the only air passages into the obstructed lung are through the collateral channels from adjacent segments, the obstructed lung becomes emphysematous and actively emphysematous in that the dilation is greater than that of maximum inspiratory filling.

There is another mechanism which works to produce progressive enlargement of lung cysts, emphysematous blebs and emphysema-

tous lung. This may be stated as follows: Whenever an air-filled cavity within the lung or a portion of lung is stretchable but inelastic, it will stretch progressively under the forces of respiration and in so doing will compress and crowd the lung which is normally elastic. It has been generally assumed that giant balloon cysts and giant emphysematous lobes have increased in size because of pressure developed within them by a ball valve occlusion of the bronchus. In the case of the giant emphysematous lobes encountered in infants no such obstruction has been found and measurement of pressure within giant lung cysts reveals that it is plus and minus zero. This means that there is a free ingress and a free egress of air. These cysts, which comprise no more than a single segment of a lobe, are small at birth. They expand to their maximum size in the first few weeks. We have seen serial x-rays of one such case in which a small cyst in the middle lobe enlarged to occupy the entire hemithorax in the course of three weeks. The actuality of this dilation by stretching can be demonstrated by employing a variation of the classical vacuum bottle representation of the mechanics of respiration. If three glass tubes are inserted through the stopper and to two of these are attached finger cots of different degrees of elasticity, when intermittent suction is exerted through the third tube the finger cot, which is the most easily stretched, enlarges more rapidly and empties less completely than that which is more elastic until in a short time it has enlarged to fill the bottle.

It seems possible, even probable, that this mechanism accounts for the giant cysts and for the giant emphysematous lobes of infants. In vesicular emphysema, once the lung portion has lost its elasticity, this mechanism undoubtedly accounts for a further increase in size of this stretchable but inelastic lung.

TREATMENT

Giant lung cysts usually represent a single segment of a single lobe. In their treatment lobectomy is the most that is usually required and in many instances these cysts can be peeled away from the adjacent segments and removed without sacrificing even a lobe.

Emphysematous blebs can be treated either by intracavitary suction or by open operation. The former method is indicated for large solitary blebs and in cases in which dyspnea is so extreme as to contraindicate major surgery. Open operation with amputation of the blebs and suturing of the pleural edges is indicated when the blebs are multiple and the patient's condition is such as to warrant major surgery.

Vesicular emphysema is rarely generalized in the sense that all of both lungs are equally involved. It is usually symmetric in any given case, either both apices or both bases being the seat of the severest disease. In some cases the remainder of the lungs are relatively normal and are functional and elastic. In such cases the dilated, inelastic emphysematous portions are a hindrance rather than a help to respiration. They compress the good lung, they limit the movement of the chest wall, they force down the diaphragm and they utilize a portion of the air exchange and the pulmonary blood flow without adding to the exchange of gases. They also cause cough. In all ways they are a hindrance rather than a help. Removal of such lobes or portions of lobes can result in an appreciable and important increase in functional vital capacity. The selection of cases is difficult and extremely important. One must be sure that he is going to increase the vital capacity rather than decrease it.

Lobectomy is rarely necessary. The extremely affected lung portions rarely comprise more than a few segments. Segmental resection, as usually performed without suturing of the lung surfaces, is not feasible in an emphysematous lung. It is our practice to place an intestinal clamp at the line at which the lung is to be resected and to run a continuous Roux suture just proximal to it. This overlapping suture is both hemo- and aerostatic. It eliminates the dangers of blood leakage into the lung and air leakage into the pleura.

SUMMARY AND CONCLUSIONS

1. Giant lung cysts, giant emphysematous blebs and vesicular and bullous emphysema localized to lobes or segments frequently produce severe dyspnea.

2. All of these conditions are amenable to surgical treatment.

3. Original theories are advanced for the progressive enlargement of these air-filled sacs and also for the pathogenesis of vesicular and bullous emphysema.

Pulmonary Air Cysts—A Therapeutic Concept

DAVID J. DUGAN, M.D. AND KENNETH L. HARDY, M.D., *Oakland, California*

THE subject of air cysts of the lung has been ably covered in the literature. It is the purpose of this paper, however, to review collectively the voluminous array of data on the topic and to relegate the mass of information to an orderly and reasonable understanding. For practical therapeutic purposes we shall attempt to classify the subject into its simplest terms. The trinity of classification, diagnosis and treatment will then form a unity of logical thought.

Because we believe that there is no acceptable surgical procedure for the treatment of diffuse pulmonary emphysema, our remarks on this topic will be concise and pertinent. We will limit our main thoughts to the cases of localized (single or multiple) pulmonary air-containing spaces in which initially, at least, the remaining lung tissue is functionally, grossly and microscopically normal.

We have chosen the all inclusive term air cyst because of its generic ability to represent all of the conditions under discussion, recognizing that its causation may be emphysematous, congenital, bronchiectatic or traumatic. We believe that this eliminates the etiologically implicating terms of bleb, bulla, congenital cyst, pneumatocele, tension cyst, etc., which have hitherto been used with free and often confusing interchange in the literature.

These air cysts may originate in any of the following four ways: (1) by extensive coalescing fragmentation of interalveolar septa resulting in an intrapulmonary emphysematous bulla in which the wall is formed by ragged adjacent lung; (2) by subpleural dissection into the visceral pleura forming an emphysematous bleb with a mesothelial wall; (3) by further development of a bronchiectatic sac which dilates from ball valve bronchial communication; or (4) by a congenital developmental arrest resulting in an air cyst with a true fibrous epithelium-lined capsule. Whether or not the etiology of an individual air cyst is due to one of the foregoing mechanisms is a moot question from the therapeutic point of view.

The presence of pulmonary air-containing spaces or air cysts carries with it the potential of four very real hazards[1] regardless of their mode of formation, namely, (1) disturbance in pulmonary function, (2) infection, (3) rupture and (4) hemorrhage. Although these dangers are modified by the nature of the air cyst in varying degree, we can, on clinical grounds alone, only conjecture as to their etiology where the remaining lung tissue is otherwise normal, and therefore must consider these threats equally in our therapeutic thinking.

A. Bleb
Bulla
Pneumatocele
Tension cyst
Giant cyst
Localized hypertrophic emphysema
Balloon cysts
Aerogenous cyst
} Air cyst

B. Etiology		Disturbance in Pulmonary Function	Infection	Rupture	Hemorrhage
Air Cyst	Emphysematous bleb or bulla	May be great	Rare	Frequent	Occasional
	Congenital cyst or grouping of cysts	Varied	Frequent	Occasional	Common if infected
	Bronchiectatic	May be great	Frequent	Rare	Frequent
	Traumatic foreign body or direct chest wall trauma)	Frequent	Frequent	Rare	Rare

With the foregoing classification in mind, purposely simplified in order to emphasize certain therapeutic implications, let us examine the possible future of these localized (single or multiple) air-containing spaces.

DISTURBANCE IN PULMONARY FUNCTION

Disturbance in pulmonary ventilation resulting from these air cysts is dependent primarily upon the nature of the bronchial communication and the state of the remaining lungs. This is succinctly brought out by Bald-

win and co-workers[4] in a careful physiologic study of sixteen cases, which they divided from the functional standpoint into three groups.

Their group I cases, in which the bronchial communication is free and patent and the remaining lungs are normal, represent a functional aberration due to dead space aeration which is roughly proportional to the size of the cyst. As can be demonstrated manometrically, these cases do not develop increased tension; but they may undergo progressive enlargement at the expense of the adjacent previously normal lung. Head and Avery's hypothesis,[19] based on direct pressures and thoracoscopic visualization, in which the cyst is seen to enlarge during expiration, further corroborates this observation. Their explanation is substantiated anatomically by the careful histologic studies of Liebow and associates,[22] who demonstrated increase in musculature of the cyst walls in emphysema at the expense of elastic tissue and vascular supply.

In Baldwin's group II cases, in which the bronchial communication is imperfect or intermittent but the remaining lungs are normal, the factor of tension may become extreme due to a one-way bronchial valve-like mechanism. Air trapping, paradoxic respiration, compression of previously normal adjacent lung, mediastinal shift and increased pulmonary vascular resistance with resultant right heart strain may then occur.

Their group III is composed of cases associated with diffuse emphysematous involvement of both lungs in which air cysts (blebs or bullae in this instance) are merely scattered exaggerations of a generalized disease. They further divide this group into (1) those with primary ventilatory insufficiency and (2) those with combined ventilatory and alveolar insufficiency. It is in the former subgroup that judicious surgical removal of a localized cyst or cysts which have undergone enlargement or tension may be extremely helpful. In the second subgroup, however, we heartily agree with Baldwin that no logical surgical therapy is available at the present time.

Disturbance in pulmonary function is of prime concern at any age, but its severity is often magnified in the infant.[3,30] Of particular interest is the follow-up on a three month old infant reported by one of us (D. J. D.) in 1947.[15] This child (Case 1) had acute respiratory embarrassment which became more severe over a two- to three-week period. Chest roentgenogram (Fig. 1) revealed a complete absence of lung markings in the left hemithorax with mediastinal shift and compression of the right lung. Needle aspiration as well as tube drainage on the left were unsuccessful. Thoracotomy was performed and a large air-containing cyst was revealed. This occupied the basilar segments of the left lower lobe and the remaining segments were normal except for compression atelectasis. The cystic area was removed by segmental resection and Figure 2 shows the x-ray appearance of the chest six years later. The child is asymptomatic and the film is considered within normal limits without evidence of residual emphysema.

INFECTION

From the standpoint of infection, certain of these air-containing spaces represent a definite threat. As has been pointed out by several workers, the tendency of air cysts due to emphysema to develop suppuration is rare,[26] although we have personally seen it occur in two cases. These patients were markedly distressed by the superimposition of pulmonary sepsis on an already impaired ventilatory situation. Both were functionally improved and also cured of their infection by conservative pulmonary resection. There is a great tendency of epithelium-lined cysts, however, to undergo suppuration. This is particularly true when their bronchial communication is poor. Many of these patients are diagnosed as having chronic lung abscess. Certainly this etiologic train of events should be considered when thinking of lung abscess, especially in young individuals who give no antecedent history of aspiration or pneumonitis.

Certainly, also, the reported cases[10,27,31] in which bronchiectatic sacs have undergone tremendous overexpansion from tension, due to the nature of the bronchial communication, are associated with chronic infection.

Pierce and Dirkse[28] reported four cases of pulmonary pneumatocele following pneumonitis. Although this is a theoretic possibility, one wonders if the actual nature of the situation was not one of suppuration in a pre-existing cyst. Perhaps the cyst, temporarily filled with exudate, was surrounded by pneumonitis and only later discharged its contents through a bronchus, the cyst then becoming apparent

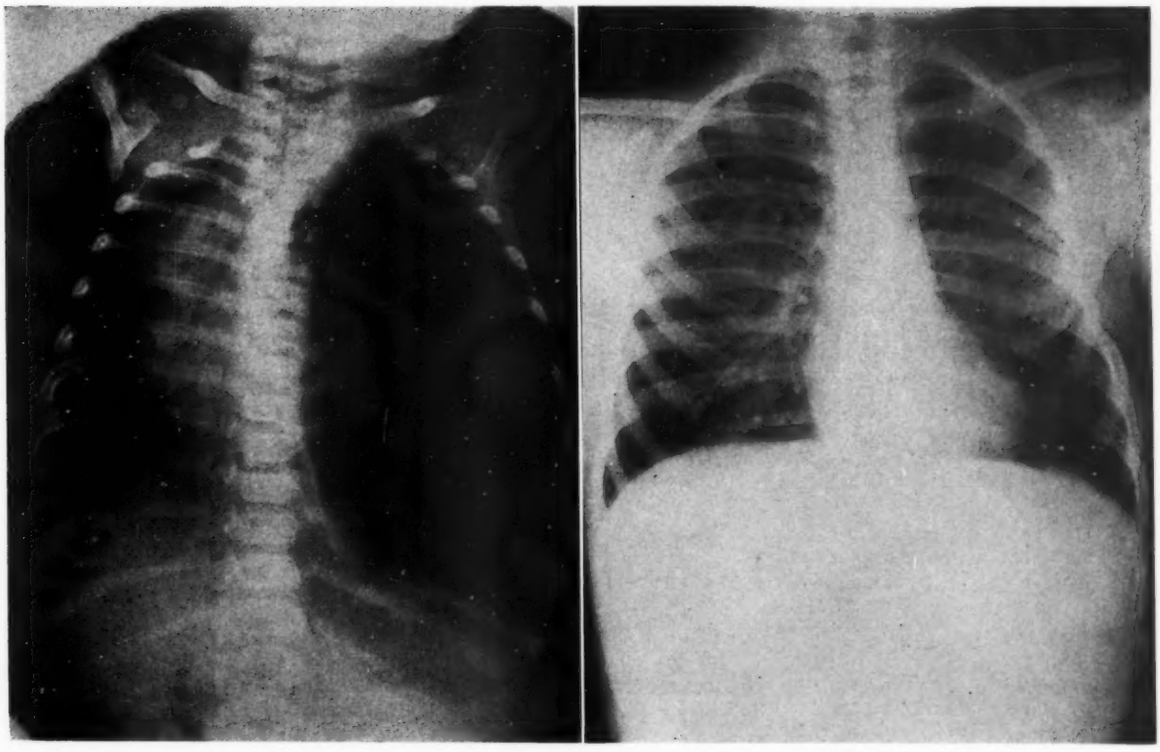

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FIG. 1. Case 1. Chest roentgenogram showing giant tension cyst occupying entire left hemithorax in a three month old infant.

FIG. 2. Same case. Chest film taken six years following roentgenogram shown in Figure 1. Note absence of residual pulmonary emphysema.

for the first time. This sequence probably occurs more frequently than we suspect.

A situation which well demonstrates the development of infection in a localized air cyst is illustrated in Case II. This forty-six year old woman had knowledge that she was harboring an air cyst in her left chest for approximately seven years. She had been under competent medical observation during this time and refused any definitive treatment in spite of progressive shortness of breath. During the seventh year of her observation she began experiencing intermittent episodes of chills, fever and cough productive of purulent sputum. Fluoroscopic examinations revealed small collections of fluid within the cyst at intermittent intervals although the fluid did not collect in any great amount. It was thought that evacuation of the cystic area was being accomplished intermittently by means of a fairly large bronchial communication. This impression was substantiated by the chest roentgenogram taken during an episode of fever and productive cough. (Fig. 3.) She then consented to surgery, and at thoracotomy a huge air cyst was found originating from the apicoposterior segment of the left upper lobe, markedly compressing otherwise normal lung. Segmental resection was performed, thereby removing the infected cyst *in toto*. The large communicating apico-posterior bronchus was closed in routine fashion and she had excellent re-expansion of the remaining lung. The surgical specimen showed inflammatory necrosis of the wall which was a pseudocapsule of compressed pulmonary parenchyma. The patient is now asymptomatic and the postoperative film is shown in Figure 4.

RUPTURE

Danger of rupture is always present in those lesions with poor bronchial communication which develop tension. Pleural symphysis is rare over these cysts,[19] and rupture is frequently and dramatically followed by tension pneumothorax and its train of disastrous events.[17,32,35] Occasionally, re-expansion of the

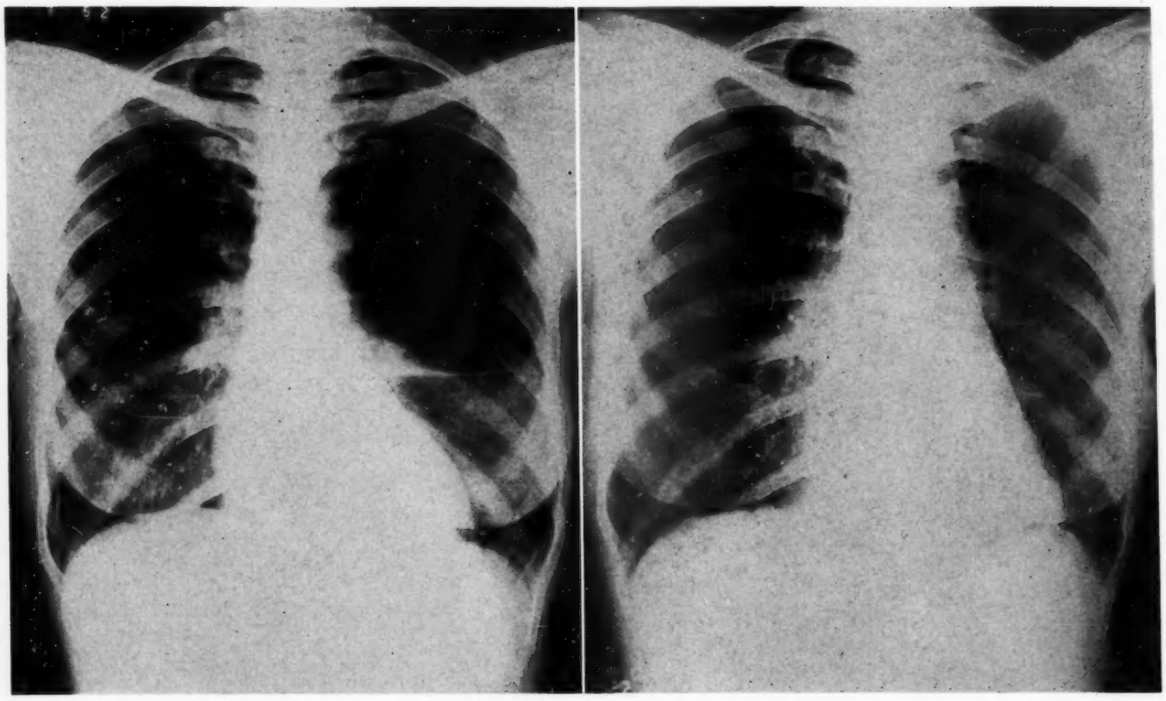

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FIG. 3. Case II. Large cystic area in left chest present for seven years. Heavy infiltration at left cardiac border indicative of secondary infection.

FIG. 4. Same case. Postoperative film following left apical posterior segmental resection for suppurative giant cyst.

lung may occur due to bronchial distortion or local tissue-flap circumstances following rupture, and normal intrapleural relationships will be restored without medical aid. It is apparent that such a fortuitous outcome cannot be predicted with certainty and the rarity of its occurrence would make its prediction therapeutically treacherous.

HEMORRHAGE

In general, the pulmonary lesions under discussion are seldom complicated by hemorrhage.[1] In the emphysematous cases, perhaps the avascularity of the cyst wall[21] itself accounts for the infrequency of this complication. The occurrence of hemorrhage is most probable after secondary infection has caused tissue necrosis and vascular erosion in the wall of the lesion. Spontaneous bleeding, however, will rarely be the cause of massive hemoptysis. Such a process may radiographically simulate lung abscess with fluid level. Spontaneous hemopneumothorax following rupture of an emphysematous bleb with persistent bleeding from a torn intercostal branch artery caused by stretching of chest wall adhesions may be extremely dramatic. Persistent bleeding from the high pressure systemic vessel in spite of conservative therapy may require early exploration.[29]

The complicating factors of rupture and hemorrhage are best demonstrated in a single case previously reported (Case III). This twenty-seven year old man experienced sudden left chest pain with dyspnea and prostration. Initial chest x-rays revealed hydropneumothorax on the left with some mediastinal compression. (Figure 5.) Thoracentesis was initially productive of 1,000 cc. of blood, and repeated aspirations over a three-day period failed to improve the situation. Because of the continuing blood loss thoracotomy was performed and a small cyst of the superior segment of the left lower lobe was found. Within taut adhesions over the cyst, a small arterial blood vessel was revealed which was responsible for the massive persistent hemorrhage. Segmental resection of the cystic area and control of the responsible vessel was followed by complete expansion of the left lung and recovery. (Fig. 6.)

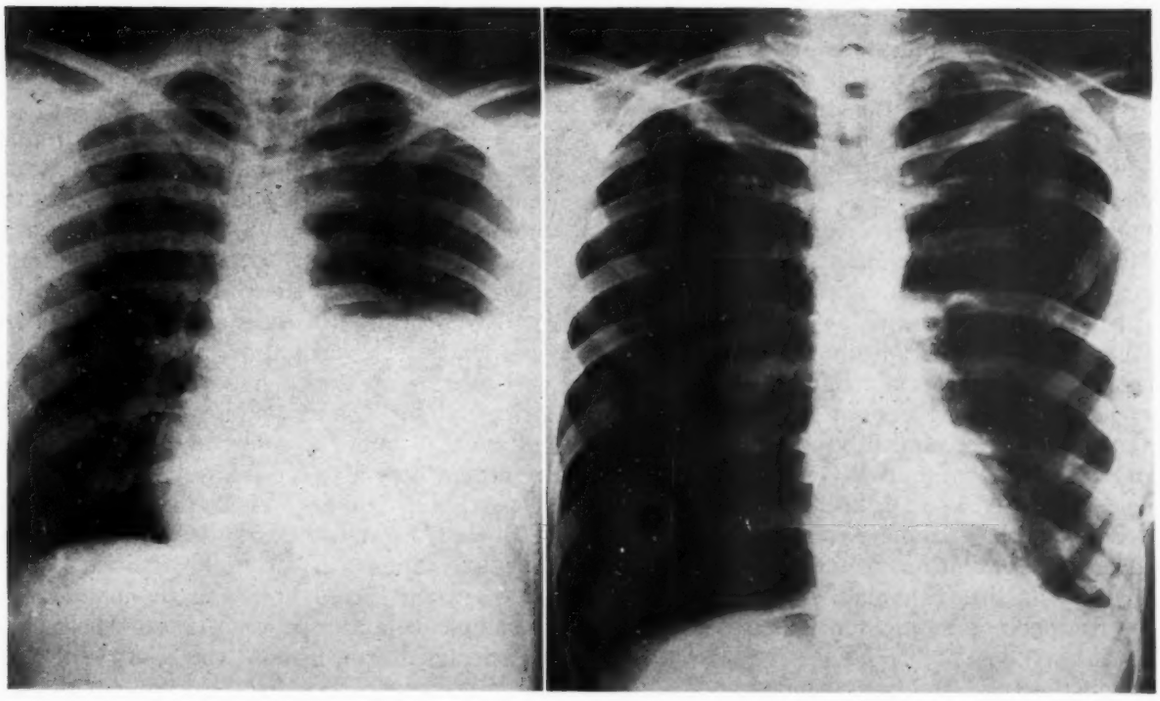

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FIG. 5. Case III. Chest roentgenogram showing hydropneumothorax due to rupture of air cyst and hemorrhage.

FIG. 6. Same case. Postoperative appearance of chest following thoracotomy for resection of ruptured and bleeding cyst.

DIAGNOSIS

The vast majority of patients with air cysts present no particular diagnostic problem. Conventional chest roentgenograms will in most instances clearly demonstrate the boundaries and extent of these air-containing spaces. It is of importance, however, to point out certain pitfalls and offer a few suggestions that might aid in more accurate evaluation of these lesions. Single small localized cystic areas disclosed during routine x-ray examinations warrant careful consideration. These asymptomatic lesions may be harmless and usually present no immediate therapeutic problem. In such cases a program of serial x-ray examinations should be pursued, having in mind surgical intervention only if progressive films demonstrate consistent or sudden enlargement. Failure to follow such cases may result in the needless sacrifice of pulmonary tissue if a progressively enlarging air space is allowed to rob adjacent pulmonary parenchyma. A demonstration of such a course of events was illustrated in one of our cases in which a program of watchful expectancy carried out elsewhere followed a relatively small air space in the right apex to the state of complete vanishing of the right lung over a period of twenty-four months. Early surgery when progression was radiologically obvious might well have saved the eventual pneumonectomy.

In contradistinction to this sequence, individual cysts may remain quiescent over many years and deserve attention in the consistently asymptomatic patient only by roentgen observation. This recommendation for conservatism in an unchanging situation presupposes that diagnostically confusing lesions[16] such as thin-walled coccidioidal, ecchinococcal, tuberculous or non-specific (possibly carcinomatous) "burned-out" abscess cavities have been excluded by studies not pertinent to this discussion. Where etiology is still in doubt, localized excision for exact diagnosis is the conservative approach.

Special x-ray technics are often helpful in some cases in which the status of the adjoining or contralateral lung is in doubt. Lordotic views for extreme apical lesions and tomographic studies for exact delineation of multiple localized cysts may be helpful. Occasionally, lateral decubitus films after the institution of

diagnostic pneumothorax may serve to differentiate pericardial and bronchogenic cysts from the group of pulmonary air-containing spaces pertinent to this discussion. The simple employment of the underexposed film as recommended by Head is frequently very helpful.

Diagnostic needling of air cysts is seldom indicated and in some instances may result in disaster.[2,6] The danger lies in the sudden rupture of the cyst wall with bronchopleural fistula, tension pneumothorax and sudden death from mediastinal compression.[20] Maier and associates have brought out the diagnostic advantage of needling fluid-filled cystic cavities when the question of empyema exists.[15] Such a maneuver is helpful, but sudden interference with the intrapleural or intrapulmonary pressure should never be attempted without adequate facilities available for immediate tube drainage or thoracotomy as the situation warrants.

Bronchoscopy has been condemned in the past on the theoretic grounds of possible rupture of a cyst due to increased intrabronchial pressure and strain. Although rupture is possible by such a means, we have not seen it occur during or following bronchoscopic procedures. The possibility of a foreign body or other remedial bronchial pathologic condition contributing to the development of the air cyst is of sufficient prominence to commend the routine use of bronchoscopy in all cases in which secondary bronchial obstruction is in question.[8]

Mention should also be made of the rare instance in which an air cyst with tension in the left lower lobe in the neonatal period may be confused with congenital diaphragmatic hernia. Multiple fluid levels and concentric air spaces in the left pleural cavity occurring in an infant should arouse suspicion as to the likelihood of diaphragmatic herniation with the presence of bowel in the thorax. Needle aspiration in such a case is definitely contraindicated and barium studies are usually diagnostic.

Fluoroscopic examination of the patient in the upright position is one of the most valuable aids in the study of these air cysts. The normal respiratory excursions may be carefully observed and the relative function of both lungs and diaphragms estimated. Air trapping, representative of ball valve bronchial communication, can be demonstrated by paradoxic mediastinal movement on the inspiratory effort. Such an observation would indicate the likelihood of continued growth of such a cyst while the absence of paradox and the diminution in the size of the radiolucent area would tend to assure its static character, for the moment at least. The Valsalva maneuver in this situation is also valuable in the fluoroscopic evaluation of these lesions.

Bronchography is of disputable worth in the preoperative investigation of an individual with an air cyst. We have found that these air-containing spaces seldom have a bronchographically demonstrable communication and its presence or absence is rarely of immediate surgical importance. When the procedure is employed, however, post-tussive and twenty-four-hour films will often show intracavitary oil when the initial films fail to demonstrate bronchial communication. It has been our experience that in general the cases which are representative of this discussion do not present associated chronic pulmonary disease, and we therefore have not used bronchography routinely in their diagnosis. If, however, in an individual case, we believe that more accurate x-ray visualization of the adjoining or contralateral lung is of importance because of the possibility of bronchiectasis, we do not hesitate to do bronchography, providing the decision for surgical intervention has been made. This then may be helpful in influencing the extent of surgical resection. Although the ventilatory interference of lipiodol and similar oily media is an objection to their use, especially in individuals with borderline pulmonary function, we have not hesitated to use these compounds if the indications as outlined heretofore were present. The objection to these agents together with the flocculent alveolar oil retention which so often persists may be overcome by the use of one of the newer, more rapidly absorbable bronchographic compounds such as dionysil.

Finally, as a further aid in the evaluation of these cases, we have employed angiocardiography as described by Miscall.[25] By such studies an estimate can be made of the integrity of the compressed adjacent lung by assaying the pulmonary vascular pattern. In certain rare instances such a study is of particular advantage; however, we believe that in most cases the careful use of fluoroscopy and routine roentgen films can give us information that is

diagnostically satisfactory, and therefore are hesitant to use the not entirely harmless intravenous radiopaque dyes routinely.

THERAPY

Conservative means of attacking these lesions have been successful in the past[14,24,33] in which needle aspiration was followed by apparent rupture and lung re-expansion. Although such results have been reported, the dangers of tension pneumothorax and its sequelae far outweigh the hazards of exploratory thoracotomy. We now believe that exploration and surgical excision is the treatment of choice.[11,30,34] As was first pointed out by Dugan and Samson in 1947,[15] those patients whose air cysts have developed tension immediately improve upon opening of the chest.[10] Formal excision of the segment giving rise to the pathologic condition in the case of a solitary cyst is undoubtedly the technic of choice. This allows for sound handling of the bronchial communication with the most lung-conserving approach. In the instance of multiple localized cysts bisegmentectomy or lobectomy, at most, may be in order. Occasionally, when multiple cysts not confined to a single lobe are found, or where contrary to preoperative evaluation the remaining ipsilateral lung is emphysematous, the local excision and plication-imbrication technic[26] is probably the most beneficial and function-sparing method. Except in rare instances, Monaldi drainage[19] or endocutaneous flap procedures[7] are obsolete in our opinion. One could conceive of a large infected cyst in a debilitated individual in which Monaldi drainage might be used as a temporary expedient. These procedures require meticulous and prolonged attention, extended morbidity, and are always complicated by infection and chronic pleural reactions not seen with modern resection technics. They were logically proposed at a time when intrathoracic procedures carried a somewhat higher risk than at the present time. The emergency use of an intracavitary catheter may be life-saving in certain instances[23] where tension in a single cyst is superimposed on respiratory impairment from bilateral generalized emphysema. We do not believe, however, that the therapy proposed by Head and Avery of a two-stage Monaldi type drainage procedure is indicated today.

A few words regarding the surgical treatment of generalized hypertrophic pulmonary emphysema[18] are germane to a discussion of this nature. We believe that in certain cases of this disease single or localized cysts which have further compromised already damaged emphysematous lung by undergoing tension warrant surgical consideration.[4,11] Their removal may be beneficial by reducing, to a degree, dead-space aeration. This presupposes that the patient has enough pulmonary reserve to survive the early postoperative period and functionally does not fall into Baldwin's group IIIB. It also implies that the removal of the offending cysts will be performed by the most lung-conserving technics possible, that is, by local excision-imbrication, segmental resection or, at the most, lobectomy. It must be emphasized that surgery in these cases is palliative, not curative, but in well chosen instances may afford the patient a higher level of exercise and pulmonary tolerance.

We do not believe, however, that any surgical procedure has yet been devised which will regenerate the emphysematous lung. We agree with Carter[9] that the problem in these patients is ventilatory and not vascular, and is most probably based on a yet unexplained loss of pulmonary elastic tissue, as emphasized by Liebow.[21] Certainly, bronchiolar obstruction fits somewhere into the picture.

A few workers have mistakenly carried over the long-established observation of an avascular *peripheral* lung in emphysema and assigned etiologic significance to it.[12,13] Although recent angiocardiographic studies have confirmed this age-old fact, the same studies have shown a marked increase in proximal bronchopulmonary vascular anastomoses in this disease. Liebow has demonstrated these anatomically, showing a tremendous increase on the venous side (bronchial vein-pulmonary vein) and a definite although lesser increase on the arterial side (bronchial artery-pulmonary artery).[22] This allows for chronic by-pass desaturation from the right to left auricles in the case of the venous anastomoses. On the other hand, the arterial anastomoses represent a conglomerate fistula which only adds to the effort of the heart already laboring under the threat of cor pulmonale. It would seem unwise to augment surgically such a mechanism.

We therefore, on theoretic grounds alone, cannot understand the proposal of advocating pleurectomy and poudrage in the treatment of emphysema. In the few instances in which

such a procedure resulted in significant systemic-pulmonary anastomoses, as disclosed by postmortem examination, it is logical to assume that the cardiac burden in those patients was thereby increased. The output and work load of the left heart was further raised to a degree proportional to the magnitude of the existing shunt plus the created shunt. There is absolutely no evidence to indicate that actual regeneration of functioning lung tissue takes place following poudrage, nor can the procedure be shown to prevent the continuing breakdown of intra-alveolar septa in the relentless emphysematous process. There is likewise no indication that systemic blood oxygenation is increased by another mechanical circuit through a lung already effete as far as alveolocapillary transfer is concerned.

We now also believe that denervation operations (autonomic ablation), procedures largely abandoned in the bronchospastic diseases, have nothing to offer in the surgery of pulmonary emphysema.

SUMMARY

Assignment of the generic term air cyst to the group of intrapulmonary space-occupying, air-containing lesions is done in an attempt to classify the subject into its simplest terms for sound therapeutic comprehension. We recognize that air cysts may be emphysematous, bronchiectatic, congenital or traumatic; but since we cannot predict their etiology with certainty until viewed histologically, their clinical and therapeutic understanding must have in mind their fate if left unattended. In the light of present-day thinking, we believe that their tendency to cause disturbance in pulmonary function, to undergo suppuration, rupture or hemorrhage, makes their surgical excision mandatory. We have indicated the applicable resection technics and have likewise stressed the futility of surgical attack in diffuse pulmonary emphysema, except in certain definite situations.

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Several patients were operated upon for hyperinsulism and no islet cell tumor was palpable. In some of these patients the body and tail of the pancreas was removed, but without clinical benefit. On reoperation usually a non-palpable, deeply imbedded islet cell tumor will be found after transections of the head of the pancreas. Johan Holst believes that islet cell tumors should be removed together with an area of surrounding pancreatic tissue, rather than to be simply enucleated. He also believes hyperinsulism without islet cell tumor must be very rare. (*Richard A. Leonardo, M.D.*)

Infected Cystic Disease of the Lung

RICHARD H. MEADE, M.D., *Grand Rapids, Michigan*

SINCE the adoption, in a widespread way, of routine chest x-rays pulmonary cysts have become commonplace. For years the literature has been full of articles on pulmonary cysts, in their various forms and with their varied complications. The related subject of infected cystic disease of the lung has received little attention. In an article published in 1953[1] the subject was discussed on the basis of nine cases in which resection had been used. It was our impression then that the condition was a common one. Since then, however, only eight more patients have been treated. These have been similar to the earlier ones, and together with them supply the material for this article.

By "cystic disease of the lung" is meant a condition in which there is replacement of normal lung tissue by multiple cysts of various sizes. Such a disease does not give rise to symptoms unless a large amount of the lung is involved. Even when there is extensive bilateral involvement there may be no definite symptoms. This is also true of simple pulmonary cysts. However, when infection of this cystic tissue occurs, the result is pneumonitis which does give symptoms; and once this area becomes infected, it rarely if ever returns to normal. An area of cystic disease is functionless. The normal mechanism for the removal of secretions is lacking. Although the cysts are usually lined with ciliated columnar epithelium, there is no apparent connection between the cystic area and the bronchial tree. The bronchioles in the area are either absent or undeveloped. Such tissue, therefore, cannot withstand the insults of infection even though antibiotics may temporarily sterilize it. The result of this infection is chronic pneumonitis, and in practically every one of our cases this was the characteristic finding. Clinically the patients showed evidence of low grade pneumonia or of chronic bronchitis. Three had definite recurrent attacks of pneumonia, whereas the others had an onset suggesting pneumonia followed by a chronic cough productive of purulent sputum, or else merely began coughing and continued to do so with production of sputum. In many cases the symptoms were insignificant and the only thing that brought the patient to us was the finding of an abnormal shadow in the x-ray of the chest. Medical treatment is of value in controlling the infection but curative therapy is limited to resection of the involved part of the lung. Of the seventeen cases in which operation was carried out, all except one recovered and have been cured of their disease. One patient, a child of three, died from pulmonary edema due to the injudicious use of intravenous fluid following the operation.

Whether or not the cysts are of congenital origin is of no clinical importance but of real academic interest. Certain solitary cysts, which are lined with ciliated columnar epithelium and have in their walls islands of tissue resembling fetal lung, are generally accepted as being congenital in origin. However, when the argument for congenital origin depends purely on the finding of columnar epithelium lining the cysts, there is reason for a difference of opinion. It is true that cystic areas may develop as the result of infection, and cystic bronchiectasis may at times present a picture resembling infected cystic disease. It is our impression that the patients upon whom we have performed resections have had congenital cystic disease which has become infected. It is not the purpose of this article to go further into this controversial subject.

COURSE OF THE DISEASE

There has been a fairly definite pattern to the disease in the seventeen patients whom we have treated. The onset has usually been described as an attack of pneumonia of rather mild degree, and generally ascribed to a virus infection. The initial illness has usually been short, and symptoms have quickly subsided after the use of various antibiotics. Complete recovery has not always occurred, the patient continuing to be tired and having a cough. When x-rays have been taken during the first illness, they have shown indistinct shadows occupying a part of a lobe. These shadows have persisted, and because of this fact that patient is known to have trouble and has been referred for study. In those instances in which there has

been complete recovery from the initial illness no x-rays have been taken. It has only been when there has been a recurrence of trouble that x-rays have been taken. If there were x-rays in the first illness, it is seen that the shadow which was present then is still present. The other patients who had an insidious onset of their symptoms have usually been considered to have chronic bronchitis and no x-rays have been taken until the symptoms have persisted for a long time. Here again it is the x-ray appearance which sends the patient in for special study. There are no characteristic symptoms, but it is rather striking that eight of fourteen adults had expectoration of varied amounts of blood at some time during their illness. None of the three children had any blood in their sputum; but as young children rarely expectorate sputum, this is not significant.

FIG. 1. X-ray of chest of patient showing shadow in left lower lobe.

DIAGNOSIS

The diagnosis of infected cystic disease is rarely made primarily. Usually the diagnosis is reached after the exclusion of other more common ailments. The diagnosis is strongly suggested when there is a history of an attack of pneumonitis, thought to be of the virus type, with x-rays showing an indistinct shadow in a part of the lung, and with clinical recovery but persistence of x-ray changes. Even more indicative of the diagnosis is the history of recurrent attacks of pneumonitis with x-ray changes persisting in the same area. Certain other diseases must first be considered and the patient studied accordingly. Tuberculosis is the first to be considered. Tuberculin skin tests and sputum and gastric washing studies must be done. If the skin test is positive, guinea pig inoculation of sputum or of gastric washings is necessary. Bronchiectasis is the disease with which infected cystic disease is most apt to be confused. Bronchoscopy for the presence of obstruction from tumor, or granulation tissue, is essential. Bronchography will demonstrate the changes due to bronchiectasis. It also will be of value at times in suggesting the presence of infected cystic disease. When bronchoscopy has failed to demonstrate any evidence of bronchial obstruction and the bronchogram reveals failure of filling of the part of the lung in which there is the unknown shadow, the diagnosis of infected cystic disease is strongly suggested. The failure of filling in such cases is due to the fact that there is no connection between the infected area and the bronchial tree. However, one cannot be absolutely sure that this is the true explanation, as obstruction of the terminal bronchi from mucus or from other material may occur. Thus it is necessary to add this bit of information to the other data. In my experience the use of iodized oil in such instances has never caused trouble but it is quite likely that the use of too much oil might be bad. The use of sectional radiography may be helpful. If the cysts are sufficiently large, they can be demonstrated by this technic. We have found this true in cases of solitary cysts but not in cases of cystic disease.

HISTORY OF TYPICAL CASE

As an example of a rather typical case of infected cystic disease it is well to review briefly the history of such a case. This patient was a child of four. At the ages of one and a half, and two and a half, he had had attacks of "bronchitis." Except for a chronic cough he had been perfectly well. His mother developed pulmonary tuberculosis and he was accordingly studied for this disease. An x-ray of the chest revealed a shadow in his left lower lobe which was thought to be due to tuberculosis. (Fig. 1.) The tuberculin skin test was negative, and studies of gastric washings were negative on smear and on guinea pig innoculation. Bronchoscopy revealed nothing abnormal. It was decided that he had infected cystic disease and operation was performed. When the chest was opened, it was seen that there

FIG. 2. Photograph of portion of lower lobe removed at operation showing the various-sized cysts.

was a mass in the anterior part of the lower lobe which felt firm. The rest of the lung was normal. This part of the lobe was removed with preservation of the large apical segment. He made an uneventful recovery and has had no further evidence of chest trouble. Examination of the specimen (Fig. 2) revealed that most of the resected tissue showed consolidation, and on section one could see multiple cysts, or abscesses, containing much greenish gray, purulent material. After evacuation of this material it was seen that the area was full of small cysts. Some were lined with smooth mucous membrane while others were lined with granular tissue. On microscopic examination the impression gained from the gross appearance was substantiated. The larger cysts were lined with columnar, mucoid epithelium which showed extensive ulceration and replacement by a considerable amount of exudate. Other areas of the specimen showed many foci in which alveoli were completely, or partly, filled with masses of polymorphonuclear leukocytes, lymphocytes and erythrocytes, as well as with groups of lipid-containing macrophages. In these areas there was an increase in fibrous tissue as well as an accumulation of erythrocytes. Bronchi and bronchioles contained considerable purulent material and there was ulceration of the epithelium.

TREATMENT

It is obvious that the only successful treatment of such a condition is excision. Ordinarily the extent of the disease is such that only partial lobectomy is necessary, but it is most important to remove all abnormal tissue. At times the remaining part of the lung may feel normal but it is without pigment, whereas the rest of the lung shows the usual evidence of anthracosis. Such tissue is almost certainly the site of cystic disease and must be removed to avoid later trouble. Even though the diagnosis of infected cystic disease cannot be made and no other definite diagnosis established, it is certainly necessary to operate upon such patients and investigate the site of the abnormal x-ray shadow. Occasionally, when all tests have been negative, the lesion proves to be carcinoma; this possibility must always be remembered. No lesion producing a persistent shadow in the x-ray of the lungs, not proved to be due to tuberculosis, should be allowed to remain in place.

SUMMARY AND CONCLUSIONS

The subject of infected cystic disease of the lung has been reviewed on the basis of findings in seventeen cases in which resection was performed. It is thought that the process is the result of infection of congenitally cystic lung tissue which, once having become infected, is unable to return to a normal state. The symptoms are not characteristic but the persistence of a shadow in the x-ray of the lung, associated with chronic cough, is always present. Tuberculosis, bronchiectasis and bronchial tumors are the diagnoses which must be eliminated first. Cure follows resection of the involved part of the lung.

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Intralobar Bronchopulmonary Sequestration

ANDRÉ J. BRUWER, M.D., *Rochester, Minnesota*

From the Section of Roentgenology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

THE association of a congenital cystic condition of a portion of the lower lobe of a lung, with an anomalous artery of supply which arises from the aorta just above or just below the diaphragm, has been termed "intralobar bronchopulmonary sequestration."

This condition is of interest to the surgeon because it represents a definite hazard to those not familiar with it. In about 3 per cent of patients operated upon death has resulted from fatal bleeding resulting from damage to the anomalous artery. In several patients the presence of the vessel was not suspected and injury to it resulted in massive hemorrhage which, fortunately, could be controlled. Injury to the anomalous artery is especially hazardous because the artery is usually quite short when it originates above the diaphragm. When it originates below the diaphragm, injury to it may cause even more difficulty owing to the danger of retraction below the diaphragm.

Embryologically, intralobar sequestration is of interest. The anomalous artery is due to persistent connections between the pulmonary plexus and the dorsal aortas, via the splanchnic plexus, which normally atrophy. When they persist, however, anomalous arteries such as those described result. Such arteries occasionally enter one or both lower lobes of normal lungs.[12] To my knowledge, the true incidence of such anomalous arteries, unassociated with other abnormalities, has not been determined at the time of this study. Of interest, however, are the cases in which a congenital abnormality of the bronchial system of a lower lobe is associated with the anomalous arteries. Some say that the anomalous artery is merely the embryonic artery of supply to the portion of lung that has been sequestered. "Except for the fact that the artery is a nutrient vessel to the sequestrated mass, their relationship is purely passive."[7] On the other hand, Pryce has suggested that the anomalous artery may be the cause of the bronchopulmonary sequestration, creating traction on and separation of a portion of the lower lobe during early embryologic development.

It is probable that extralobar bronchopulmonary sequestration (inferior accessory lung), various degrees of pulmonary agenesis, certain types of diaphragmatic hernia[5,6] and some types of pulmonary vascular anomalies[18] have, broadly speaking, a similar embryologic basis. Such a proposal was implied by Cole and coworkers based on the work of Needham; they suggested that "The most likely explanation is that some interference with the embryologic organization takes place in the lower chest behind the heart and that all structures under development in that area, at the time may be affected." Although diffuse and intangible, this hypothesis is basically no doubt the most accurate.

INCIDENCE

Although the literature contains reports of necropsy in fewer than ten cases of intralobar bronchopulmonary sequestration, it contains descriptions of surgical procedures in almost 100 cases.[1,4,9,15–17] The surgical significance is indicated by the fact that there have been three deaths at operation owing to injury to an unexpected anomalous artery. The majority of lesions have been on the left side.

SYMPTOMS AND DIAGNOSIS

Although the condition is discovered sometimes from routine roentgenograms of the thorax made during surveys or annual checkups, the majority of patients come to operation because of repeated acute pulmonary episodes associated with intermittent cough, occasional blood-streaked sputum, fever, chills and roentgenographic evidence of a pathologic process in one or the other lower pulmonary fields.

The roentgenologic findings are strikingly similar in most cases. If the sequestered seg-

ment has no communication with the bronchial tree, the cysts are filled with mucoid material. On a roentgenogram of the thorax made from the postero-anterior view such a cyst, if large enough, shows as a rather well defined solid mass, whereas the lateral view shows a less well defined misty outline. Wyman and Eyler have pointed to the fact that the long axis of the lesion sometimes appears to be directed posteriorly and toward the midline, as if indicating the possibility of a connection with the aorta in the region of the diaphragm. If a communication with the bronchial tree exists, the lesion may contain one or more air-fluid levels. Fry and his colleagues[7] have made the interesting suggestion that even when a communication with the normal bronchial tree does not exist, the lesion may contain a pneumatic area overlying the fluid, and that this is due to "metabolic gas."

Although the roentgenographic appearance of the lesion in itself is not diagnostic, the finding of a lesion in the characteristic location, usually in a young person who is either symptomless or who gives a history of repeated attacks of pneumonia, makes bronchopulmonary sequestration an outstanding diagnostic possibility. Familiarity with the clinical picture and roentgenographic features of intralobar bronchopulmonary sequestration has enabled some physicians to diagnose the condition before operation.

PATHOLOGIC ASPECTS

The gross abnormality usually falls into one of the three groups of Pryce: (1) a large cyst in the sequestered region, (2) a polycystic mass, or (3) a sequestered mass which contains dilated bronchi that branch parallel to the anomalous artery. In any of these gross types the bronchial or cystic elements may be completely dissociated from the bronchial tree or there may be a small communication with the bronchial tree, perhaps as a result of infection. Kergin, for example, thought that in four of his five cases the connection of the cystic region with the bronchial tree was secondary. One also may find that in a specimen with many cystic regions some may have small communications with the bronchial tree while others will be totally isolated and filled only with a jelly-like substance, probably inspissated mucus. The cysts are lined with respiratory epithelium. Alveoli in the tissues surrounding the cysts and pigment in the involved pulmonary tissue are not evident; both would have been expected if any functioning pulmonary tissue had been present. It would appear that the conducting component of the involved region (that is, the bronchial component) is abnormally developed and expanded into cystic structures, whereas the functioning (alveolar) component is undeveloped. The tissue between the expanded bronchi is largely fibrotic and contains a variable number of inflammatory cells, depending on the amount of infection that has occurred. Infection of the sequestered lung tissue presumably may take place by contiguity from an adjacent, temporary infection of the lung, or by hematogenous spread, as probably occurred in one case reported by Pryce.

In most instances the anomalous arteries are confined to the sequestered region. As a rule veins are not found to accompany the anomalous arteries and it is presumed that the venous drainage from the sequestered region is via the normal pulmonary veins. Kergin has pointed out that in some cases this may result in a significant arteriovenous shunt. The anomalous artery usually enters the lung in the region of the lower limit of the pulmonary ligament. It is not unusual for large lymph nodes, which may be devoid of pigment, to occur in the region of this accessory hilus. Although these anomalous arteries have been referred to as bronchial type arteries, Price has pointed out that they have the histologic appearance of true pulmonary arteries. Not infrequently considerable intimal sclerosis is present, a feature which some authors consider to result from the high pressures in these vessels, but which also may be owing to the fact that they are essentially functionless and that their natural tendency is therefore to deteriorate.

CASE REPORTS

Case 1 is of special interest because of the patient's long clinical history, age and the early roentgenograms which were still available. (Fig. 1.)

CASE 1. A man, sixty years of age, presented himself at the Mayo Clinic in 1951 with the chief complaint of a long history of cough, productive of about one-half cupful of yellow sputum per day. Recently he had lost weight and appetite, and had severe malaise as well

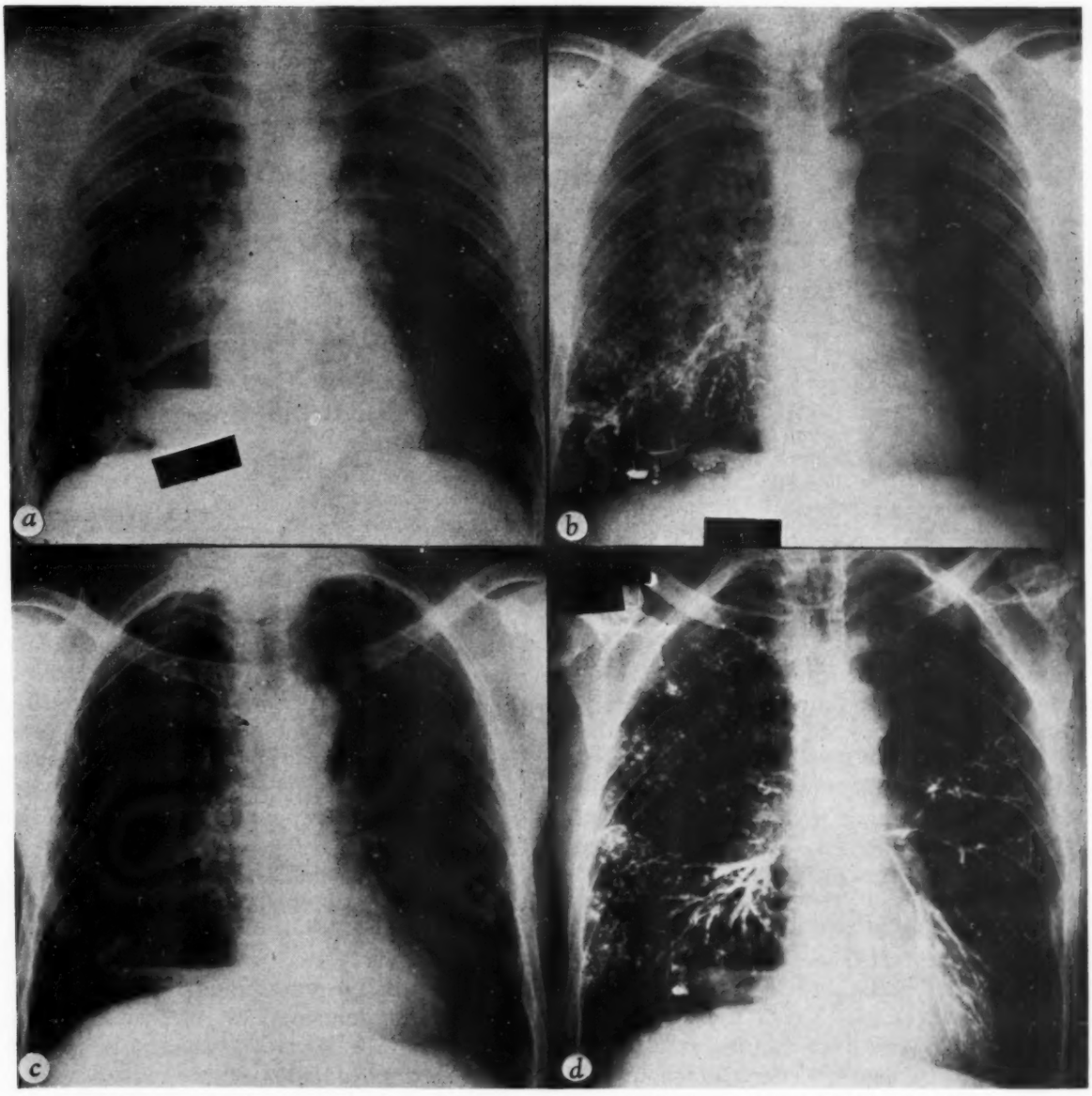

FIG. 1. (*a*) Postero-anterior view taken in 1928. Cavity in base of right lung with air-fluid level. (*b*) Bronchogram made in 1928 showing evidence of bronchiectasis near the cavity. (*c*) Postero-anterior view taken twenty-three years later, in 1951, shows the lesion to be essentially unaltered. (*d*) Bronchogram made in 1951 shows some increase in the bronchiectasis surrounding the cystic lesion. Opaque material has not entered the cyst in spite of the fact that the air-fluid level indicates that some communication with the bronchial tree must have been present.

as dyspepsia. He had not had night sweats, fever or hemoptysis. Twenty-three years previously the patient had come to the Clinic because for six years he had had spells of coughing, productive of large amounts of pus. At that time, in 1928, a roentgenogram of the thorax revealed evidence of a cavity with fluid level in the right lower lobe, in the posterobasal segment, and bronchograms revealed evidence of associated bronchiectasis in this region. (Fig. 1*a* and *b*.) A great deal of pus was aspirated through the bronchoscope. Phrenicotomy was performed and the patient was relieved considerably for a number of years. Roentgenograms and bronchograms (Fig. 1*c* and *d*) of the lung in 1951 appeared remarkably similar to those made twenty-three years previously. Examinations of the sputum for tuberculosis, actinomycosis and other fungi gave negative results. At operation a long band of indurated tissue extended toward the midline from the medial basilar portion of the right

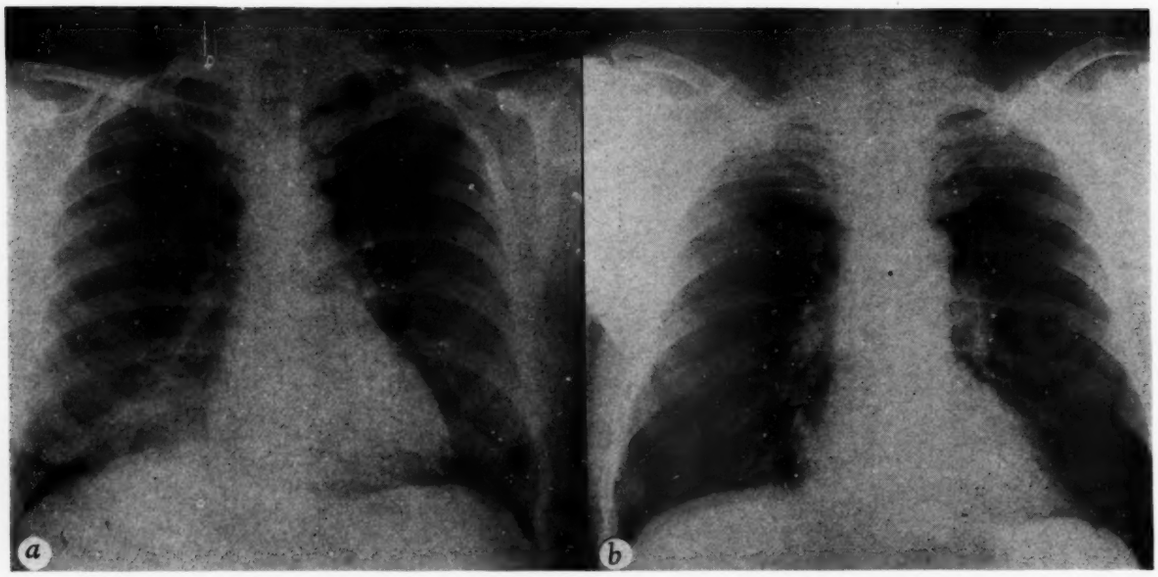

FIG. 2. (*a*) Case II. (*b*) Case III. Postero-anterior views in two cases of intralobar bronchopulmonary sequestration. Wyman and Eyler drew attention to the obliquity of the long axis of some of these lesions, suggesting the possibility of vascular attachment to the lower thoracic or upper abdominal aorta.

lower lobe. This band contained an artery, 0.8 mm. in diameter, apparently coming off the lower part of the thoracic aorta. A cystic structure was felt in the lower lobe, and this part of the lung was very adherent to the diaphragm and lateral thoracic wall. The right lower lobe of the lung was removed. A cyst, 6 cm. in diameter, lined with columnar ciliated epithelium, was present in the posterobasal portion of the lobe. The anomalous artery, which was very sclerotic, ramified around the periphery of the cyst. Some of the surrounding lung tissue was bronchiectatic.

CASE II. A woman, thirty-six years of age, who had been in excellent health, came to the Clinic because a mass had been found in the thorax on routine roentgenographic examination. A roentgenogram made at the Clinic showed evidence of a well circumscribed mass in the posteromedial part of the base of the right lung. (Fig. 2*a*.) Exploratory operation revealed a cyst in the posterior part of the right lower lobe associated with a few adhesions. An anomalous artery from the lower part of the thoracic aorta entered the pulmonary ligament of the right lung. The lower lobe of the right lung was removed and the patient made an uneventful recovery. The artery was found to ramify in the region of the cyst.

CASE III. A fifty-seven year old man presented himself at the Clinic for evaluation of backache. On routine roentgenogram of the thorax evidence was shown of an oval mass of homogeneous density in the posterobasal region of the right lower lobe. (Fig. 2*b*.) The long axis of this oval density appeared to be directed toward the midline, a feature described by Wyman and Eyler.[19] Bronchoscopy gave negative results as did studies for tuberculosis, fungi and brucella.

At operation dense adhesions were found in the posterior costophrenic sulcus in the posterobasal region of the lung. A large anomalous artery was found in these adhesions. Considerable difficulty was encountered in ligating it, owing to its tendency to retract toward its origin from the lower thoracic aorta after it had been divided.

The right lower lobe was found to contain a trabeculated bronchogenic cyst containing chocolate-colored material. Surrounding it and in the sequestered region were many small bronchial structures, but no evidence of alveoli. A communication between the cyst and the normal bronchial tree was not apparent. The anomalous artery was very sclerotic.

CASE IV. Although in excellent health, a twenty-two year old nurse had had several roentgenograms of the thorax in the last five years which had shown evidence of a lesion in the base of the right lung. A roentgenogram made at the Clinic in December, 1953, revealed evidence of multiple rounded densities, each about 3 cm. in diameter, in the posterior basal

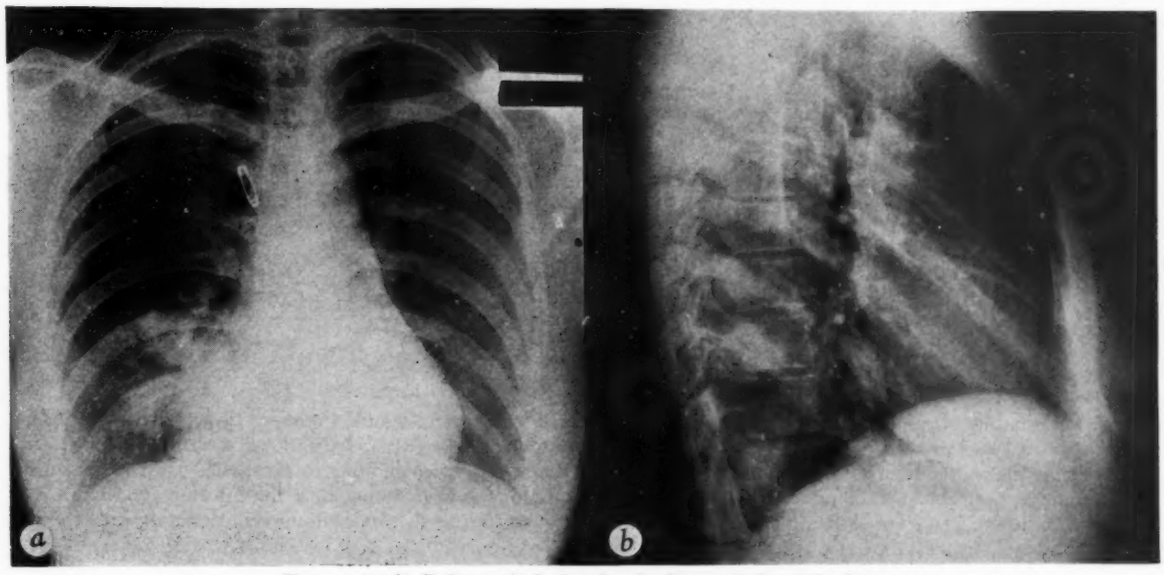

FIG. 3. (*a*, *b*) Polycystic lesion in the base of the right lung.

segment of the right lower lobe. (Fig. 3*a* and *b*.) Roentgenograms made in the patient's home town in 1949 were obtained and showed the same lesions, but the densities were slightly larger in those made in 1953. Suggestions as to the nature of the lesion included multiple granulomas, arteriovenous anomaly, hamartomas, metastatic process and bronchiectatic abscesses. The surgical consultant suggested multiple bronchogenic cysts and warned about the possibility of an anomalous vessel. Results of all other preoperative studies were negative.

At operation the right upper and middle lobes were found to be normal. The posterior basilar segment of the right lower lobe contained numerous doughy-feeling cysts. An artery about 1 cm. in diameter arose from the aorta immediately above the diaphragm and entered the right lower lobe through the inferior end of the pulmonary ligament. The right inferior pulmonary vein drained normally. No other vascular or bronchial abnormalities were present. Lobectomy was performed on the right lower lobe. The patient made an uneventful recovery.

The pathologic specimen contained multiple cysts from which a thick, reddish brown material was aspirated. The anomalous vessel entered the region of sequestration. Pathologic sections were not made because it was decided to do injection studies with the use of liquid plastics. This aspect of the case will be reported in the near future.[11]

COMMENT

Intralobar bronchopulmonary sequestration is of interest in that the rather typical clinical findings in a young person and the presence of a "solid" lesion, or a cystic lesion with an air-fluid level in the region of the posterior basal segment, make the diagnosis a good likelihood. It is thus possible for the roentgenologist to draw the attention of the surgeon to the possibility of encountering an anomalous artery in the posterior basal region.

In Cases I and III the patients were much older than the usual patient having intralobar sequestration. Case IV is unusual in that multiple large cysts were present.

Indicator dilution studies might provide evidence of early recirculation through the lung, as in the case of peripheral arteriovenous fistulas.[2]

In view of the similar origin of anomalously draining pulmonary veins[8] and of anomalous arteries, we have been interested in finding abnormalities in the bronchial tree among patients having anomalous venous drainage of a lung.[3] In one case in which the right lung drained into the inferior vena cava, the bronchus of the right upper lobe apparently had failed to develop. (Fig. 4*a*, *b* and *c*.) The venous anomaly was proved by cardiac catheterization. In another case (Fig. 5*a*, *b* and *c*) with similar roentgenologic findings of anomalous venous drainage of the right lung, a major bronchus, probably that to the lower

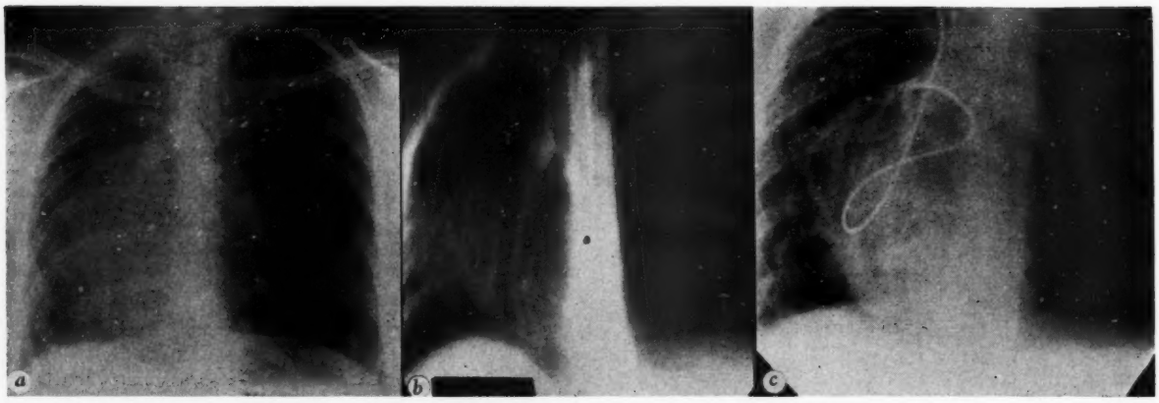

FIG. 4. (*a*) Shadow of anomalous vein projected behind cardiac outline. The vein courses downward toward the diaphragm and was proved at catheterization to enter the inferior vena cava. Note dextroposition of heart and the fact that the right hemithorax is smaller than the left. Bronchography demonstrated agenesis of the bronchus to the right upper lobe. (*b*) Tomogram demonstrating anomalous vein draining into the inferior vena cava. (*c*) Selective angiopneumogram showing small right pulmonary artery and the anomalous vein draining into the inferior vena cava.

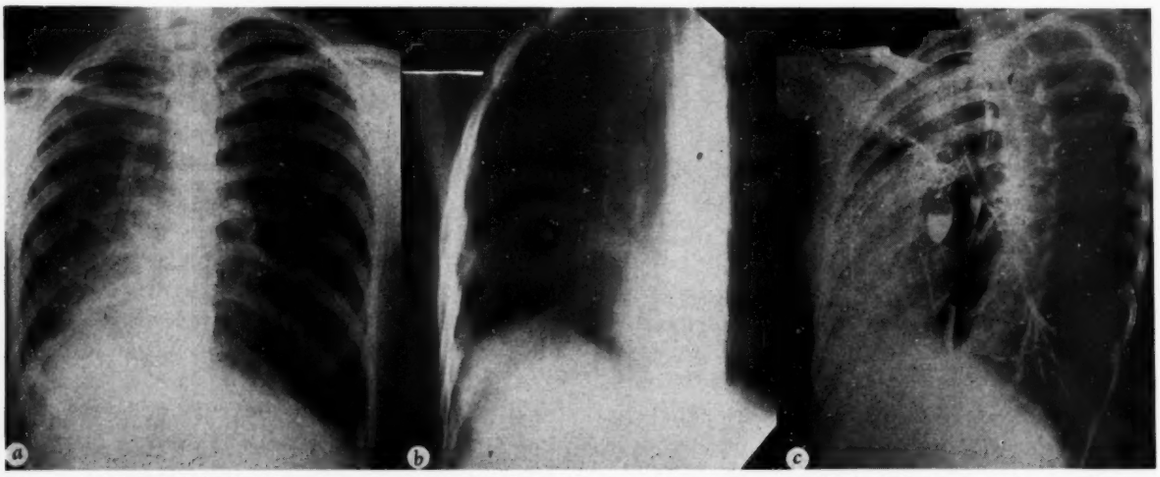

FIG. 5. (*a*) Small right hemithorax as compared to left. Abnormalities in this case included dextroposition of heart, cystic hypogenesis of the bronchus to the right lower lobe and the presence of an anomalous vessel. (*b*) Tomogram in same case showing anomalous vessel and bronchial abnormality. (*c*) Bronchogram showing cystic hypogenesis of right lower lobe bronchus.

lobe, ended blindly in a cyst-like structure. Such findings as these lend further support to the previously quoted statement of Cole and co-workers regarding the pathogenesis of the condition.

SUMMARY

Intralobar bronchopulmonary sequestration is the association of a cystic lesion in the lung, practically always in the posterior basal segment of a lower lobe, with an artery of supply arising from the lower thoracic or upper abdominal aorta. This artery is of surgical significance, as it may offer a definite hazard during lobectomy.

The anomalous artery is the result of persistence of connections between the dorsal aorta and pulmonary plexus via the splanchnic plexus. Such an artery may exist alone or in association with a number of developmental abnormalities such as pulmonary cysts, diaphragmatic hernia, arteriovenous pulmonary fistula or eventration of the diaphragm.

Intralobar sequestration usually manifests itself clinically in young adults. Patients have a history of repeated attacks of upper respiratory infection, and roentgenograms of the thorax show evidence of a cystic lesion in the posterobasal segment of one of the lungs.

Attention is drawn to the similarity of em-

bryologic development of anomalous pulmonary veins, and to the occasional association of abnormalities of the bronchial tree with anomalous venous drainage of the lung.

Indicator dilution studies would be interesting in the diagnosis of intralobar sequestration.

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Hydatid Cysts of the Lung

LOUIS R. DAVIDSON, M.D., *New York, New York*

IT is generally agreed that the lung, after the liver, is the organ of man's body which is most frequently infested with hydatid disease. This condition is the result of the development of the cystic or larval state of the tapeworm, Taenia echinococcus. Hydatid cysts of the lung were clinically known to the ancients. In modern times considerable impetus to the study of this tapeworm disease of man has been afforded by the work of Dévé, the eminent French authority, the studies of Harold Dew of Australia and the writings of Jose Arce, the Argentinean surgeon. These include, among other matters, the different manifestations of hydatid cyst such as metastatic and secondary hydatid disease. The geographic distribution of this condition "is dependent on its incidence and distribution in reservoir hosts, particularly sheep, cattle and pigs."

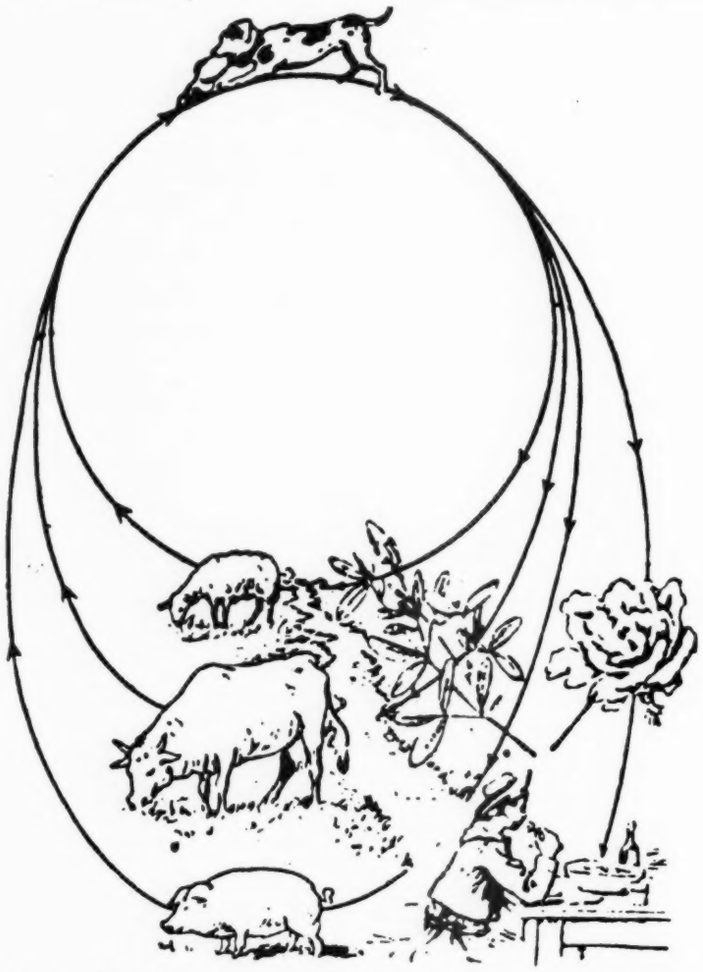


FIG. 1. Depicting the natural history of the Taenia echinococcus. (From VEGAS, M. H. Hydatid Cysts of the Lung in Children. Buenos Aires, 1928. Imprenta Lamb y Cia, Ltd.)

Native cases are uncommon in this country but they are far from infrequent in sheep-raising countries such as Argentina, Uruguay,

New Zealand, Australia, Italy, Syria, Greece and Algiers.

A hydatid cyst can grow in the human and yet remain symptomless. Cysts of the lung, of course not palpable, are revealed almost exclusively by roentgenograms which, as in other pulmonary lesions, are most important in the diagnosis of the condition.

FIG. 2. Taenia echinococcus, showing all segments. (From DAVIDSON, L. R. Hydatid cyst of the lung. *J. Thoracic Surg.*, 13: 471–512, 1944.)

The development (Fig. 1) of the cyst in man is readily understood if a few brief moments are given to the study of the evolution of the taeniae (adult state) and the larva. Of course where there are sheep, there are dogs. The infected dog (also the wolf and jackal) is the definitive host; in its small intestine worms or taenia (Echinococcus granulosus) are found. The intermediate or reservoir hosts are many, but most commonly infested are the sheep, pigs and man. Man is an "accidental host as far as the further biological development of the parasite is concerned; human infestations represent a dead end" (Dew). Man, then, does not take part in the continuation of the cycle. The untainted dog is infested by ingesting the organs of animals, usually the sheep, which have been afflicted with fertile hydatid cysts (containing scolices). One notes that the hydatid cyst in man is the result of the development of an ovum, or ova, in one or more organs of man. The ovum has its origin in the T. echinococcus which resides as a parasite in the dogs' intestine. This worm is very small. (Fig. 2.) Its length is about 0.5 cm. A chain of segments forms the entire tapeworm. It is composed of a head, neck and two segments or proglottids. The terminal compartment is gravid and it is from this, the broadest segment, that eggs are discharged. The head is provided with four suckers and is armed with many hooklets by which the taenia fasten themselves to the intestinal mucosa of the dog. An infested animal contains many hundreds or thousands of worms; in turn these taenia discharge into the feces innumerable ova which gain entrance into man (the intermediate host) usually through contaminated water or vegetables.

When the egg lodges in man's stomach, the gastric juice digests the external coat thus freeing the hexacanth (six-thorn) embryo. (Fig. 3.) Subsequently, it migrates through the gastric wall or the duodenum entering the venules lodging in the first barrier, the liver. If this shelter is hurdled, the embryo passes through the inferior vena cava into the right side of the heart and then is filtered out of the blood stream by the lung.

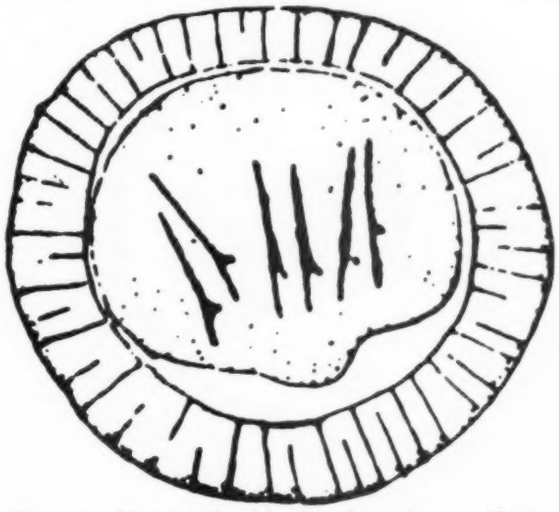


FIG. 3. Hexacanth (six-thorn) embryo. (From DAVIDSON, L. H. *J. Thoracic Surg.*, 13: 471–512, 1944.)

Let us consider that it remains and develops in this latter organ (Fig. 4) and there assumes what is called the larvated stage. The embryo in growing empties its center. The vacuolation continues so that at a certain point one finds a small vesicle which contains fluid. The expanding embryonal wall is composed of two zones: the inner nucleated wall (germinal layer) and the outer non-nucleated wall (laminated layer). From the former wall masses of cells which become vacuolated protrude

into the vesicle's cavity. These capsules form the proliferative vesicles or brood capsules which in turn give rise to the scolices. These are the heads or the source of new worms. Most of these capsules and scolices are pinched off the germinal layer and sink in the hydatic fluid (aqua de roca, crystalline fluid), becoming what has been called "hydatid sand." The fluid-containing bag, composed of two layers of hydatid sand (Fig. 5A and B), is the hydatid vesicle. As the vesicle, which is the parasite, grows, the organ in which it resides reacts to its presence forming a perivesicular layer or adventitia or pericyst. Obviously this is formed by the host. The connective tissue layer which surrounds the hydatid vesicle forms, with the latter, the hydatid cyst. The adventitia embraces the hydatid vesicle. However, it is not united to it. This holds true in the simple cyst. The adventitia bears the same relationship to the vesicle that the banana skin has to the fruit. Let us say this cyst developed in a sheep instead of man; further imagine that the infested organ of this slaughtered animal is eaten by a dog. The scolices are ingested and enter the dog's small intestine where they become attached to the mucosa developing into T. echinococcus. Obviously the entire cycle is repeated. Nothing will be mentioned about daughter cysts which occasionally develop, except to indicate that they are usually the result of a mechanical, infectious or toxic influence and usually they are biologically sterile, that is to say, they are without scolices or proliferative vesicles. On the other hand, a fertile cyst, if ruptured or aspirated, may discharge scolices (Fig. 6) into the surrounding tissue, thus causing the origin of new cysts which are known as secondary cysts (secondary hydatidosis). This leads to the obvious truism that when operating one must not permit any of the hydatid fluid or hydatid sand to contaminate the incisional or operative wound. In addition, for this reason, among others, one should never aspirate a hydatid cyst except at the time of operation.

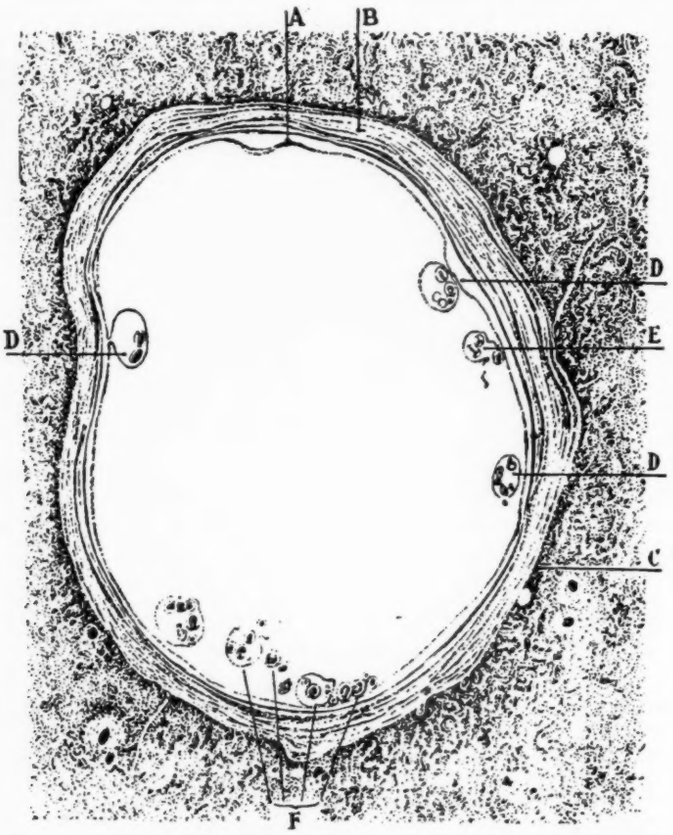


FIG. 4. A, germinal layer; B, laminated layer; C, adventitia; D, proliferative vesicle; E, scolices; F, hydatid sand. (From ARCE, J. *Arch. Surg.*, 42: 3, 1941.)

Again, if the primary cyst erodes into a vessel, metastatic hydatidosis results.

The main difference between primary and secondary cysts is that the former develops from the ova and the latter grow from the scolices.

The fluid and the membranes in the simple cyst are bacteriologically sterile. Of course, cysts without scolices and brood capsules are biologically sterile and are called acephalocysts. Simple hydatid cysts may assume a new phase and become complicated. When this occurs, the adventitia becomes infected or damaged, thereby altering the integrity of the germinal and laminated layers along with the contents, with resulting death of the vesicle. In consequence of this the cyst is usually septic and the hydatid vesicle virtually falls apart. The contents are necrosed and fragmented.

Susman has very pithily discussed the biologic features and their relationship to the "incidence of the disease and its distribution in different organs." I take the liberty of quoting him verbatim:

"1. The ova are hardy and resistant; the scolices are delicate and labile. In practical terms this means that the intermediate host may become infected long after the ova have been excreted by the dog; but the dog will become infected only if he eats the scolex-bearing cysts while they are still fresh and unruptured.

"2. Only a small percentage of human beings, dogs, and sheep are susceptible to infection.

"3. Many ova that reach the intermediate host fail to develop into cysts and many cysts abort at an early stage.

"4. The distribution of cysts in the body varies from country to country and from species to species. In Australia and New Zealand about 23 per cent of the cysts in human beings occur in the lungs; in Iceland the percentage given by Icelandic investigators is only 1.5 per cent. Cattle have proportionately more pulmonary cysts than sheep, and pigs have fewer. Cysts are rare in the horse, and a very high percentage of them occur in the liver.

"5. The parasite may die at any time for no apparent reason, or as the result of rupture or pyogenic infection. It may become a caseous mass very like a tuberculous focus.

"6. The rate of growth of these cysts is not known. It has often been said that most patients become infected in childhood although the disease may not be diagnosed until adult life. But radiology has shown that a hydatid cyst may become apparent in a matter of months; if infection did actually occur in childhood in these persons, then for many years the cysts were too small to cast any appreciable shadow and then they suddenly took on rapid growth. Furthermore daughter cysts may appear in radiograms taken during the first year after rupture of a mother cyst. It is probable that primary and secondary cysts have different rates of growth and the rate of growth varies from animal to animal, as well as in the small animal.

FIG. 5A. Membranes extracted from patient. (From DAVIDSON, L. R. *J. Thoracic Surg.*, 13: 471–512, 1944.)

"7. It is not known when and where the ova hatch out. But we do know that the embryos enter the portal circulation and that explains why most hydatid cysts are found in the liver. One or more embryos may pass through the liver and reach the lungs which are the second most frequent site of cysts. Again some embryos pass through the lungs and enter the general circulation. Thus cysts may develop in any part of the body, but the total number in other organs and tissues is much less than the number in the lungs and liver. Cysts in different parts frequently coexist.

"8. Hydatid cysts grow by quiet pressure necrosis of surrounding structures and any tissue may become seriously eroded, including bones and blood vessels."

In the lung the parasite develops into a hydatid vesicle with its surrounding adventitia (Fig. 7); the latter is covered by a thin con-

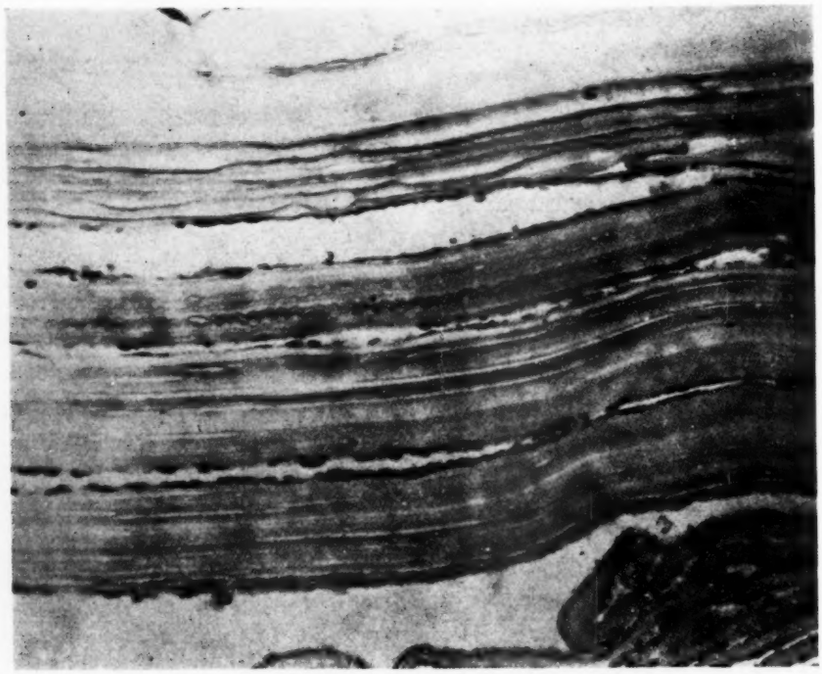


FIG. 5B. Germinal layer and laminated layer. (From DAVIDSON, L. R. *J. Thoracic Surg.*, 13: 471–512, 1944.)

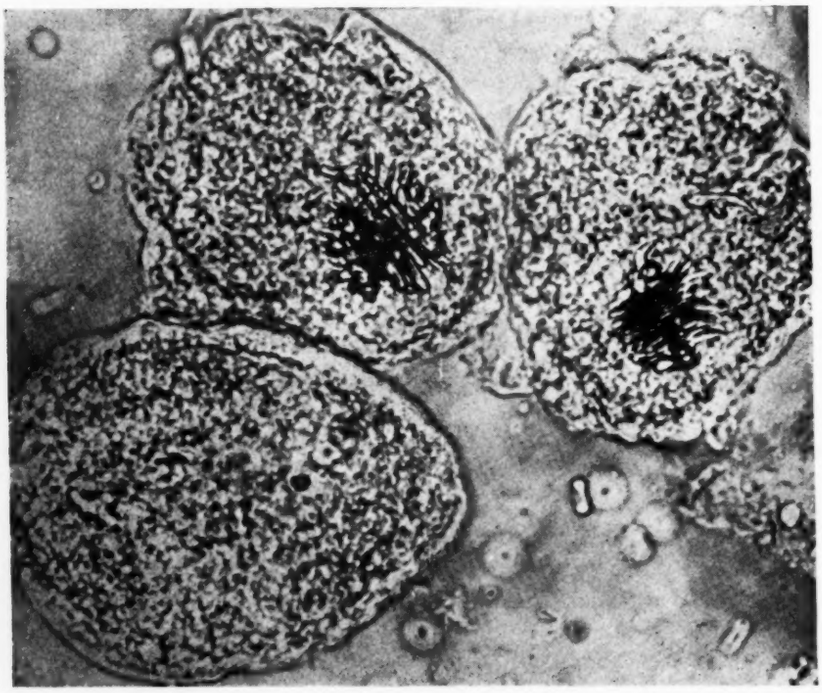


FIG. 6. Scolices with cupping. (From DAVIDSON, L. R. *J. Thoracic Surg.*, 13: 471–512, 1944.)

centric layer of atelectasis. The enlarging cyst, through an adventitial defect, impinges upon a bronchiole whose wall, in turn, may contact the outer surface of the laminated layer. As the cyst increases in size, the bronchiole is eroded thus permitting the perivesicle space to communicate with the bronchiole. Now through various acts such as coughing and straining, air insinuates itself between the "banana skin and fruit." Then more air surrounds the vesicle causing the perivesiculum pneuma. In most instances further changes are noted. If the membranes tear, fluid enters the bronchiole and the act of coughing is stimulated. If a larger opening occurs in the membranes, air enters the vesicle and a double-dome arch, Cumbo's sign, is produced. Finally, when the echinococcus membranes sink into the cavity and float upon the remaining fluid, the camalote formation is noted. This was first described in 1924.[4]

It is necessary, for the understanding of the symptoms and signs, to recognize the difference between simple and complicated cysts which may be either peripheral or central. The simple or ordinary cyst generally is silent. Cough is usually the first symptom and, of course, is non-productive. Occasionally there is companion chest pain. Later the cough may be productive and accompanied by hemoptysis. Later, when the cyst enlarges, dyspnea may manifest itself. This symptomatology does not reveal the possibilities of an exact diagnosis. The physical examination often suggests the diagnosis, but of course it is not unmistakable. "In countries with echinococcus-infested zones, the place where the patient lives is of the utmost importance."

Diagnosis is difficult at the best. Laboratory methods are quite valuable, yet these procedures are not unequivocal. Eosinophilia is not present in dead or suppurating cysts. Even in intact cysts the positive blood picture is not always present. Writers differ, yet one may state that eosinophilia is present when the percentage of eosinophils is over 4 per cent. Of course, other conditions produce this picture. If a high eosinophilia count is obtained, one or the other of the biologic reactions which are about to be described should be present before a diagnosis is made. The complement fixation test (Weinberg) is not invariable. Of all, the intradermal test of Casoni is most reliable although it is not constant. Certain diagnosis is made only when hooklets (Fig. 8), scolices or laminated membranes are found. However, in complicated cysts one can make a positive diagnosis when a characteristic x-ray picture is found noting the perivesiculum pneuma, Cumbo's sign, or camalote formation.

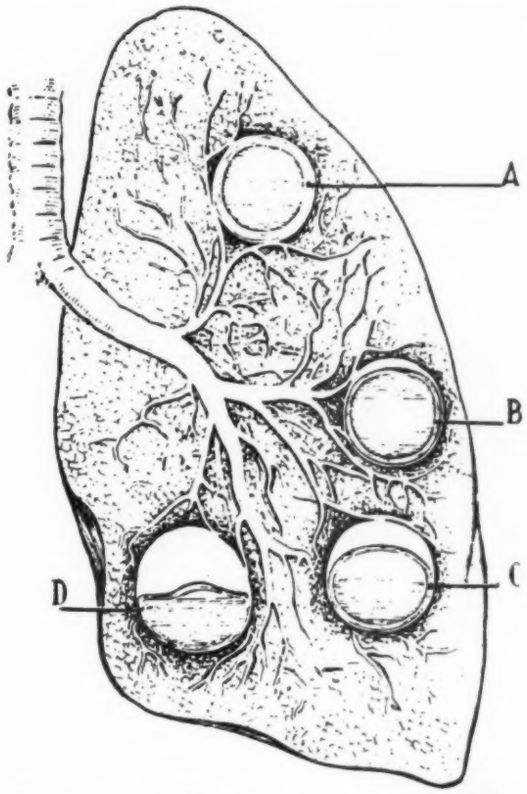


FIG. 7. A, hydatid cyst of lung; B, perivesicular pneuma; C, double-dome arch (Cumbo's sign); D, camalote formation. (From ARCE, J. *Arch. Surg.*, 42: 11, 1941.)

In the following case a preoperative diagnosis was made simply upon the basis of such an x-ray.

M. N., a white adult man who was working as a ship's steward, was born in Portugal and had lived in Argentina a part of his early life. In May 28, 1937, at the time of his first admission to the United States Marine Hospital at Stapleton, Staten Island, New York, he was thirty-five years of age. One month before, while on board ship, he was struck by a swinging door and suffered a lacerated wound of the back. Two days later pain developed in his left chest. On June 3rd he spat blood. An x-ray picture of the left upper lobe revealed a dense circular shadow "the size of a hen's egg." A diagnosis of a superior sulcus tumor was made.

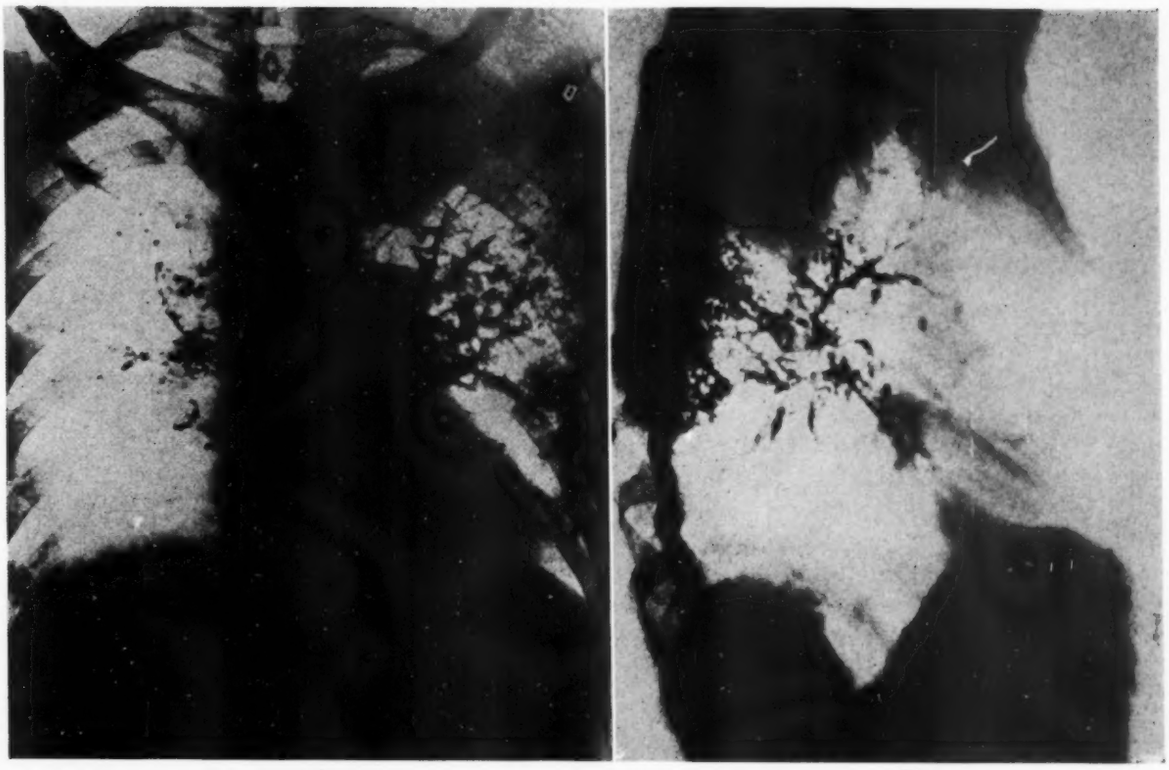
FIG. 8. Hooklets from scolices. (From DAVIDSON, L. R. *J. Thoracic Surg.*, 13: 471–512, 1944.)

His second admission was March 10, 1939, when he entered the Marine Hospital because of an infected right leg. Again a chest x-ray disclosed the same shadow as that noted earlier. There had been no weight loss, night sweats, fever, cough or hemoptysis. On this occasion an additional diagnosis was made by the x-ray department, that is, fibroma of the lung or a benign mass.

On March 14, 1941, the patient was again seen because of a blow suffered in the left temporal region. Of course, the chest mass was noted again; it was stationary and "not typical of tuberculosis." On April 2nd bronchoscopy was done again and bronchograms made. All these examinations were negative.

It was learned that this patient had been admitted to Ellis Island Hospital as far back as September, 1934, at which time he had had cough, fever and blood-tinged expectoration. At no time had the tubercle bacillus been found. The x-rays always showed the circular shadow in the apex of the left upper lobe.

His last admission into the United States Marine Hospital at Stapleton was November 12, 1941, at which time he was suffering from pain in the left side of the chest, hemoptysis and cough. The sputum was negative for tumor cells. On fourteen different occasions his sputum was negative for the tubercle bacillus. At this point I saw the patient for the first time. X-rays were presented which permitted an immediate and unequivocal diagnosis (Fig. 9A and B) of a complicated hydatid cyst. On January 30, 1942, operation was performed. A section of the left fourth rib was removed and the soft tissues were draped. The lung was adherent to the parietal pleura. The cyst was opened and suction was used. Much degenerated tissue was obtained and remnants of structures which looked like "empty grape skins" were extracted. The defect was packed with gauze soaked with 5 per cent formalin. Later the gauze was removed and dry gauze packing was introduced. The wound was dressed every other day. The patient was discharged on April 16, 1942, with no cough or febrile reaction. The longitudinal incision was healed. X-rays showed complete healing of the lung. The patient gained 20 pounds in weight. The pathologic report indicated the presence of mucopurulent putty-



9A 9B

FIG. 9. A, frontal view; again note perivesicular pneuma. B, lateral view; note perivesicular pneuma. (From DAVIDSON, L. R. *J. Thoracic Surg.*, 13: 471–512, 1944.

like material. Here and there, there were cheese-like masses. There were no hooklets, laminated membrane or scolices. The patient is at this time working as a seaman.

If the hydatid cyst ruptures into the pleural cavity, grave consequences may ensue; an anaphylactic shock manifests itself quite rapidly. However, it may be lessened or relieved by the use of adrenalin. Anaphylaxis is obviously the result of the absorption of the antigen in an individual who has been sensitized by the development of the hydatid vesicle in the lung.

Medical treatment is unavailing. Puncture of the cyst, whether for the purposes of diagnosis or treatment, is mentioned merely to be condemned. It follows then that surgery is the only treatment. What cysts should receive intervention and when should operation be performed? There is some difference of opinion. However, one must ask, In what group is the cyst? Is it simple or complicated? Central or peripheral? Simple cysts offer no symptoms; occasionally there is a slight unproductive cough with some spitting of blood. If the parietal pleura is contacted by the cyst, there may be some pain. Complicated cysts frequently reveal pathognomonic x-ray signs. As in a putrid lung abscess, the presence or absence of pleural adhesions determines the type of surgical therapy. Regardless of the presence or absence of pleural synechia, the essence of treatment is identical: The chest wall is opened, the adventitial wall is incised and the membranes are removed. A one-stage operation in the absence of adhesions is neither simple nor without a definite risk. Central cysts should not be attacked unless they are draining poorly and are accompanied by fever. Even then caution is suggested.

Lobectomy may be indicated for serious or recurrent hemoptysis, intensive complicating bronchiectasis, a centrally located cyst or a markedly infected cyst.

The chest wall opening is made over the site of the peripheral cyst which previously has been revealed by x-ray. With the presence of adhesions between the visceral and parietal pleura definitely established, and after the parietal pleura has been exposed, the adventitia is entered and the membranes and contents are

removed. If when the parietal pleura is exposed the lung is found unadherent, the surgeon may produce adhesions by packing the wound with gauze. About two weeks later the packing is removed and the adhesions will be noted between the visceral and parietal pleura. Then the adventitia is incised and the vesicle extracted. On the other hand, a one-stage operation is conducted by projecting the lung through the wound in the parietal pleura. This is accomplished through the use of a tight-fitting gas mask, at the appropriate moment the pressure in the bag being increased to about 10 cm. of water, positive pressure. Another type of one-stage operation for the unadherent lung is performed by transfixing the lung, pleurae and part of the chest wall with sutures outside of the cyst area. Thus the lung is held to the chest wall, preventing a large pneumothorax. This procedure is not looked upon with favor.

The cyst itself is treated in different ways. Of course, the wound should always be meticulously draped before the cyst wall is opened. After an exploratory needle has been thrust into the cyst, fluid is withdrawn. Some complicated cysts have no fluid but this is usually known before the operation. Some operators inject 2 per cent formalin into the cyst after the fluid has been aspirated. In any event, the pericyst is incised and the membranes are extracted from within the adventitia. In a simple aseptic cyst the adventitia may be completely sutured. In most cysts the pouch (adventitial membrane) should be marsupialized and drainage established. Septic complicated cysts may be repeatedly packed with gauze until healing has been established.

Hydatid cysts have been found in practically every organ of the human body. Fortunately the cyst is usually single, although on rare occasions two or more cysts are found and sometimes in one organ or part. Multiple organ infection occurs in less than 2 per cent of the cases. As in bronchiectasis, so in hydatid disease, the infestation usually first manifests itself in children. In man the liver is involved in about 75 per cent of the cases; the lung is infested in about 10 per cent of the patients studied. Peculiarly, and in contradistinction to this, in the squirrel the lung is involved in 98 per cent of the cases and the liver in 0.2 per cent (Dew). This is said to be in consequence of the fact that the hepatic capillaries of this animal are readily enlarged, thus permitting the parasite to filter through. In addition, some biologic peculiarity rather than circulatory effect may influence the site of the lodgement of the embryo.

All hydatids begin as simple cysts; but in the lung when the cyst enlarges, it involves a bronchus or bronchiole into which it leaks and from which the dissecting air is obtained. The contents may be evacuated through the bronchus, permitting a spontaneous cure. On the other hand, the cyst may be contaminated and all fertile elements are destroyed. The scolices die and the membranes are destroyed.

Daughter cyst formation is rare in the lung and as already noted it is the result of some complication.

G. T., a thirty-eight year old laborer, was born in Syria and came to this country at the age of ten. He was admitted to the Post-Graduate Hospital on September 25, 1940, with complaints of inability to use his lower extremities, decrease in sensation of these extremities and tumor in the left chest wall.

Twenty-three years before he had suffered from recurrent abscesses over the sacral and lumbar regions. They were allegedly tuberculous in nature. They did not heal for some time and then only when he exposed the parts to direct sunshine. Six years before admission a painless, barely discernible mass was noted in the chest wall on the left side, posteriorly. Three years before admission he complained of pain in this area and was x-rayed by eminent Philadelphia radiographers who diagnosed his condition as a tuberculous process. Almost two months before admission he first complained of loss of sensation in the lower extremities. One morning shortly thereafter he could barely walk. The patient could not flex his legs. He said that "they were dead." There were no urinary or gastrointestinal disturbances.

The patient was a well nourished, robust, young adult man whose gait was severely impaired. A non-tender mass was noted on the left side of the chest, mainly in the posterior axillary region extending from the sixth to the tenth ribs. Over the sacrum there were depressed pale scars. There was bilateral weakness of the legs, the left more marked than the right; the knee and ankle jerks were absent. There was a bilateral Babinski sign but no bladder or bowel disturbance. Below the fourth lumbar vertebra there was some patchy hyper-

esthesia. A spinal tap was negative. The spinal Wassermann and the blood Wassermann and Kahn tests were negative.

X-rays revealed cystic-like degenerative changes with marked expansion involving the ninth rib on the left side and invasion of the neighboring soft tissues with encroachment on the pulmonic field with pleural retraction. There was a left paramediastinal mass. There was erosion of the seventh rib and invasion of the posterior 2 inches of the eighth rib. The lesion also invaded the left transverse processes of the arches of the seventh, eighth and ninth dorsal vertebrae. There was no eosinophilia. A working diagnosis was made of either a benign tumor undergoing malignant changes or an extensive tuberculous process. It was decided to perform a biopsy. On September 28th the first operation was performed, consent having been obtained for a biopsy only. A tumefaction was found particularly in the area of the ninth rib over which an incision was made. The chest wall muscles were barely opened when some fluid appeared in the wound. Upon enlarging the wound an amazing number of daughter cysts accompanied by fluid and hydatid sand were disgorged to the surface. The ninth rib was found to be destroyed, and the seventh and eighth ribs eroded. More surprisingly, however, a multiloculated supra- and infracostal cyst of definite proportions was found. Aspiration of the contents was performed and the wound was packed.

On October 19th the previous incision was enlarged and extended, passing through the back muscles. The cavity was almost ruthlessly and completely unroofed using, in the main, large biting rongeurs. After the wound was saucerized, cleaned and painted with formaldehyde, a large leg roll was packed into it.

The pathologist reported daughter cysts, laminated connective tissue and scolices. The specimen consisted in the main of dense fibrous scar tissue with lymphoid and plasma cell infiltration. The hydatid fluid was bacteriologically sterile.

Fracture boards were placed beneath the patient's mattress; a Knight brace with a chin and occiput extension was worn. He did receive a short period of galvanic stimulation to the lower extremities. Postoperatively intradermal and complement fixation tests were negative. For some time before his discharge (February 6, 1941) he had been up and walking about the ward, although he had been admitted to the hospital in a wheelchair.

On January 6, 1943, he was readmitted to Post-Graduate Hospital, still wearing the spinal brace. At this time he had no ankle edema although this condition had been present at the time of discharge. On examination two discrete, though not too well defined, palpable fluctuant masses were noted over the left erector spinae muscle at about the level of the ninth dorsal vertebra. On January 9th these cysts were operated upon, being isolated and aspirated. The walls were removed intact. The area was packed with 10 per cent formalin gauze. About three days postoperatively this patient, who had gained considerable weight and was now definitely obese, developed a rash which was thought to have resulted from the formalin packings. These were discontinued. The x-rays disclosed much improvement, particularly good fusion of the dorsal vertebra. At the time of this communication he was walking well, eating well and feeling better than he had in many years. He was working as a laborer and was not wearing the brace.

In this connection, it is proper to relate that the acceptance of this case, in which daughter cysts were found, as one originating in the lung must be questioned. Of course, superficially at least its picture is that of an intrathoracic hydatid cyst. Dew has stated that "whenever an intrathoracic cyst is found to contain daughter cysts, an hepatic origin should be suspected and this should lead to an investigation of the diaphragmatic region." However, we are not sure but that this cyst did not originate in the vertebral column. Possibly an implantation first appeared in the pedicle, body or lamina of the vertebra. This presumably began when the patient was a boy in Syria. Many years later the soft tissues were invaded by extension. Just before he was seen, cyst formation within the spinal canal reached such a stage that pressure was exerted upon the cord and concordant symptoms appeared. This, of necessity, is a very slow process because decompression probably had occurred time and again by extraspinal extension. Of course, the accuracy of the observations made at the time of the operation may be questioned. The history, findings and postoperative neurologic improvement must permit of the biting inference that this was not a pulmonary

hydatid but rather it was a hydatid cyst originating in the spinal column.

Again, according to Dew, "the prognosis in vertebral hydatid disease is almost hopeless as regards complete cure. This is due to the impossibility of removing by surgical means all diseased bone . . . to the multiplicity of osseous cysts, to the certainty of recurrent pressure on the cord and to the risks of infection." It is also noted in this case that at the time of the operation when unsuspected scolex containing fluid was evacuated, these premature heads were undoubtedly implanted in the operated wound thereby permitting of the growth of a recurrent or a secondary hydatid cyst.

Almost all authorities deprecate the aspiration of the hydatid cyst. In many instances this procedure has been followed by death. Even if death does not result, infection of the cyst and/or contamination of the pleural cavity are probabilities. Even if the foregoing do not occur, there is always the possibility of creating the presence of a lifeless foreign body in the lung. In fact, the death of the parasite does not remove the patient from the possibilities of danger. Finally, let it be said, it takes many years to learn whether leakage around a puncture needle occurred with subsequent implantation of scolices in the chest wall.

It is not unusual to have a simple cyst leak and rupture into a bronchus or bronchiole. If this progresses, the fluid and the membranes may be extruded, permitting a spontaneous cure. If only the fluid is expelled, the membranes remain and one may expect an infection or abscess formation. Paradoxically, these complicated cysts may possess one virtue, that is, a pathognomonic radiograph may be obtained. As already indicated, a preoperative diagnosis was made in view of the presence of a perivesicular pneuma. This feature has been most adequately described by Arce and Zehbe.

There is but one treatment of hydatid cysts and that is surgical. The absence or presence of adhesions modifies the plan of attack. If there are definite adhesions, the rib overlying the cavity is removed and the contents are in turn extracted.

Most authorities insist, particularly with a simple cyst, that one should never think that the adhesions are either quantitatively or qualitatively what one desires. Therefore the operation should be performed in either two stages or, if in one stage, the lung should not be permitted to retract into the thoracic cage. This is prevented by using increased anesthetic gas or oxygen pressure. Finally, the localization is very well noted by a spot-shot. It follows then that the impression is that a pulmonary hydatid cyst should be operated upon in two stages or in one stage using increased anesthetic pressure of 10 cm. of water when the cyst is approached.

Estevez discusses the injection of 2 per cent formalin into the cavity after it has been punctured and aspirated. Dévé most emphatically insists that this medicament should never be used in the lung or pleura. Of course, under all circumstances both the incisional wound and the pleural cavity should be well protected during the operation. The aspirating unit must be functioning and immediately at hand. Fluid is not always obtained from a complicated cyst. After the cyst has been removed, the adventitia may be closed tightly or it may be sutured to the chest wall bringing a drain from the pulmonary defect (marsupialization).

Biologic reactions are encountered in many ways. As in all helminthic diseases, eosinophilia is noted. This is not a constant finding. The Weinberg test, which has been confirmed, is a complement fixation test. One must possess a fresh antigen properly tested. Although more reliable than the leukocyte count, it too is not always reliable. The best type of test is that which involves an intradermal reaction and is known as the Casoni test. A positive reaction (like a Mantoux test) presents a wheal and a surrounding area of erythema.

The diagnosis of pulmonary hydatid cyst is difficult, particularly in the United States where it is a rare condition. Since this cyst becomes complicated quite often, certain differences arise which add to the problem. In brief, one may be suspicious of a case if there is a history of cough, pleural pain, hemoptysis, and/or a febrile reaction, particularly if the patient has been an inhabitant of some infested area. X-rays may be of great help and other laboratory findings as indicated may be helpful. Cysts usually are in the lower lobe and occur more frequently in males.

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In the days before antibiotics and other modern improvements it was understandable why many surgeons preferred conservative surgery in the treatment of sigmoidal diverticulitis. However, now that resection of the colon can be performed with so low a mortality, Lewis and Hurwitz believe that excision of the involved sigmoid should be the recommended procedure, not only because it is more certain to effect a cure but also because in the doubtful case there might be a malignancy instead, and radical excision is the only procedure with a chance of cure. (*Richard A. Leonardo, M.D.*)

Pulmonary Arteriovenous Fistula

CHRISTOPHER J. STRINGER, M.D., ARTHUR L. STANLEY, M.D., RICHARD C. BATES, M.D. AND JOHN E. SUMMERS, M.D., *Lansing, Michigan*

WHEN arteriovenous fistula is mentioned, one immediately thinks of Doctor D. C. Elkin and arteriovenous fistula of the general circulation.[39–50,53,58,88,111,137] However, arteriovenous fistula of the lesser (pulmonary) circulation is something entirely different from arteriovenous fistula of the greater (systemic) circulation. Arteriovenous fistulas of the systemic circulation most frequently result from penetrating wounds of war and operative trauma and, to a lesser degree, are of congenital origin. The vessels most commonly involved from trauma are popliteal, femoral, subclavian and axillary, brachial, tibial and peroneal, those of the head and neck and the radial and ulnar vessels.[122] Arteriovenous fistulas of the greater circulation may follow surgery. The first report of an arteriovenous fistula was made in 1762 by William Hunter and consisted of two cases which followed injury to the brachial vessels after bloodletting operations.[47] In 1946 Elkin[47] reported a case of arteriovenous aneurysm of the uterine vessels following hysterectomy. He also found in the literature reports of arteriovenous fistula following operation for ruptured intervertebral disk; intercostal arteriovenous fistula following thoracentesis; of the facial vessels due to application of a Roger Anderson splint; of the anterior tibial vessels following introduction of a Steinmann pin; of the superior thyroid artery following thyroidectomy; in amputation stumps and in the renal vessels following nephrectomy. Arteriovenous fistula of the phrenic vessels following thoracentesis,[45] arteriovenous aneurysm of the vertebral vessels following wounds[48] and arteriovenous fistula between the middle meningeal artery and the greater petrosal sinus following skull fracture[53] have been reported. Eight hundred fourteen aneurysms and fistulas resulted from World War II,[53] and 106 such lesions have so far been reported from the Korean War.[122] The local and systemic effects of arteriovenous fistulas of the greater circulation have been extensively studied.[50] A continuous murmur is heard over the lesion. Aneurysmal dilatation of the involved vessels may occur. (Aneurysm of a vessel must be differentiated from aneurysmal dilatation of an arteriovenous fistula.) The superficial veins distal to the fistula become dilated. The area distal to the fistula may show evidence of deficient circulation. The artery and vein proximal to the fistula dilate. All collateral vessels dilate. The systemic effects of an arteriovenous fistula of the systemic circulation are: increase in the blood volume, increased cardiac output and increase in the size of heart. These systemic changes are seen in about 50 per cent of the patients and depend upon the size of the vessels involved.[137]

Arteriovenous fistula of the lesser (pulmonary) circulation differs from that of the greater circulation, first as to etiology. Since pulmonary arteriovenous fistula is a congenital lesion and since it is a variant of congenital hereditary hemorrhagic telangiectasis, it is desirable to consider the latter disease first.

CONGENITAL HEREDITARY HEMORRHAGIC TELANGIECTASIS

In 1876 Legg described a patient with numerous small telangiectases on the face, with painful swelling of the joints and a history of epistaxis. Epistaxis had occurred in three generations of this family.[56] Chiari in 1887 reported on a family with a history of epistaxis occurring in four generations, and with multiple telangiectases of the skin and mucous membranes.[56] Rendu in 1896 was the first to associate the tendency toward epistaxis with telangiectasis occurring in families as a clinical

entity.[106] In 1901 Osler[100] presented three cases of recurring epistaxis associated with multiple telangiectases of the skin and mucous membranes, and stated that Babbington and Rendu had previously reported cases. Osler[100] wrote: "The condition has nothing to do with haemophilia, with which the cases had been confounded. . . . Angiomata are very peculiar and remarkable structures, in which I have been interested for many years." He described three types of angiomas: (1) the pinpoint, punctiform or capillary angioma; (2) the solid, nodular nevus; and (3) the spider angioma.

In 1909 Hanes[68] introduced the term "hereditary haemorrhagic telangiectasia." He defined the disease as "a hereditary affection manifesting itself in localized dilatations of the capillaries and venules forming distinct groups of telangiectases which occur upon the skin of the face, nasal and buccal mucous membranes, and give rise to profuse hemorrhage, either spontaneously or as a result of trauma." The telangiectases usually do not appear before the age of puberty. In the third and fourth decades of life they become more numerous and more subject to hemorrhage. The telangiectases are bright red in color; and as they enlarge with venule participation, they become violaceous. Hematemesis, hemoptyses, melena, hematuria and cerebrovascular accidents may occur. Thus the diagnosis of hereditary hemorrhagic telangiectasis is facilitated by: (1) the hereditary tendency toward the disease; (2) the presence of typically distributed telangiectatic areas; and (3) the tendency for the telangiectatic areas to bleed.[79]

Hereditary hemorrhagic telangiectasis is sometimes referred to as "Osler's telangiectasis" and "Rendu-Osler-Weber disease."

In 1950 Garland[56] reviewed 218 families with hereditary hemorrhagic telangiectasis and added twenty new families; later[57] he added three more families with the disease. In 238 families he found 648 males, 703 females and sixty-four of unstated sex who were afflicted. The defect appeared to be transmitted by males and females in equal proportions.

The disease has been observed in families of most of the European races, and appears to be relatively more common in Jews than in Gentiles. It is more common than is generally realized. Many patients with this defect are treated for epistaxis without the cause of their symptoms being appreciated.[56]

PULMONARY ARTERIOVENOUS FISTULA

Churton[26] is credited as being the first to describe pulmonary arteriovenous fistula, although he did not appreciate the nature of the defect, calling the case "multiple aneurysm of pulmonary artery." The complete report is as follows: "Doctor Churton showed the lungs from a boy 12 years of age in whom a loud roaring pulmonary systolic bruit and very highly accentuated second sound were present during life, with haemoptysis, epistaxis and dropsy. Four of the secondary branches in one lung, and three in the other, led to aneurysms as large as walnuts, filled with blood clot. The boy had been ill for a year. He had never been strong; had bronchitis at the age of 4 years, and again at 6; pleuritic effusion in July 1895 and was subsequently said to have Bright's disease. In April or May 1896 he had three attacks of haemoptysis; ascites appeared; face became swollen after this. He died in July. There were several wart-like growths, firm and smooth, on the tricuspid valve and the endocardium near it."

In 1917 Wilkens[143] gave a complete description of the clinical and autopsy findings of a sixteen year old girl with multiple pulmonary arteriovenous fistulas, but he also called it "a case of multiple pulmonary aneurysms." Wilkens[143] described the epistaxis ("oft Nasenbluten"), the cyanosis ("leichter Zyanose der Lippen"), the dyspnea ("Gelindes Asthma"), clubbing of fingers and toes ("Trommelschlegelfinger und zehen") and the telangiectases of the lips and tongue ("Punktformige Gefasserweiterungen an Lippen und Zungenspitze"). He described murmurs in both axillas, and the chest x-ray showed densities in both lung fields. Five years after the first hospital admission the patient was readmitted with intense cyanosis and died three weeks later at the age of twenty-three. At autopsy the left pleural sac was filled with blood (over 1,500 cc.). Wilkens demonstrated by dissection and injection multiple pulmonary arteriovenous fistulas of both lungs, one of which had ruptured into the left pleural cavity. It is also to be noted that this patient had hereditary hemorrhagic telangiectasis.

The first case of arteriovenous fistula of the lung diagnosed clinically was reported in 1939 by Smith and Horton.[127] The patient was a forty-seven year old white man who was seen in 1938 with complaints of attacks of numbness,

dizziness, convulsions and exertional dyspnea. He had been a "blue baby." Cyanosis and clubbing of the fingers and toes had been noted at twenty-three years of age while he was in the army, and again at forty years of age at the Mayo Clinic. He had been diagnosed as having polycythemia rubra vera and was treated with phenylhydrazine and by venesection without benefit. Chest x-ray revealed a lesion in the base of the right lung, and a continuous murmur was heard over the lesion. Blood count: red blood cells 6,470,000, hemoglobin 23.7 gm. Angiograms were performed revealing that the density in the right lung was vascular in nature. The diagnosis was arteriovenous fistula of the right lung.

Pulmonary arteriovenous fistula, from an etiologic standpoint, should be called "cavernous hemangioma." It has also been termed "pulmonary angioma" and "pulmonary arteriovenous varix or aneurysm." It should not be confused with pulmonary aneurysm, as in simple pulmonary aneurysm there is no fistulous shunt between a pulmonary artery and vein.[69] It is now clear that pulmonary arteriovenous fistula is a variant of hereditary hemorrhagic telangiectasis.[56,57,63,68,73,78–80,97,100,139] The hemangioma in the lung may vary from a small capillary hemangioma which obviously could not give clinical symptoms to extensive bilateral cavernous hemangiomas causing death due to the arteriovenous shunt. A few instances of capillary hemangiomas of the lung have been reported. DeLange and DeVries Robles in 1923 reported the case of an infant who prior to death at two and a half months of age had had numerous convulsions, and a diagnosis of brain disease or meningitis had been advanced. At autopsy he was found to have two capillary hemangiomas about 1.5 cm. in diameter in the left upper lobe, and one capillary hemangioma 3 cm. in diameter in the right lung. "The tumor consists of massive new growths of small and smallest capillaries in some places overfilled with blood . . . all of the tumors show the same structure." In 1940 Plaut[104] reported two cases of capillary angioma of the lung. The first case was a thirty-five year old woman with the typical clinical symptoms of pulmonary arteriovenous fistula, and with cyanosis and clubbing of the fingers and toes; autopsy showed numerous intravascular angioendotheliomas of both lungs. The second case was that of a fifty-three year old woman who died of heart disease. At autopsy a small 3 by 2 mm. capillary angioma was found in one lung. This latter lesion was found incidentally and was too small to cause any appreciable arteriovenous shunt in the pulmonary circulation. Plaut[104] reviewed the slides of the reported cases of DeLange and DeVries Robles,[31] and Case 3 of Edwards and Taylor,[38] and noted that they were all similar microscopically (capillary hemangiomas).

In this series (Table 1) are included all known cases (two) of malignant hemangioma originating in the lung. Malignant hemangioma is very rare, only eight cases having been reported.[109] In 1914 Wollstein[146] reported the case of a four month old baby girl who had been a "blue baby" at birth. The infant had exhibited cyanosis, clubbing of the digits and dyspnea. Autopsy revealed a large solid hemangioma of the right upper lobe, cutaneous hemangiomas and generalized metastatic hemangiomas to the abdominal viscera. In 1935 Hall[67] presented the case of a forty year old woman who at autopsy presented massive hemorrhage in the right pleural cavity from a large hemangioma in the right lung. True metastases consisting of both cavernous and capillary cellular areas were found throughout both lungs, the pleurae, retroperitoneal lymph nodes and the liver.

Generalized benign hemangiomatosis may occur. In 1929 Jaffe[74] described the case of a baby girl who died at nine months of age. Autopsy revealed abscess of brain and streptococcal meningitis, abscess of the left thigh and bilateral bronchopneumonia, a large hemangioma of the side of the neck, about 100 small hemangiomas of the skin, multiple hemangiomas of the leptomeninges, the anterior roots of the cervical nerves, dura mater spinales, the diploë of the parietal bone, the thyroid, the lungs, the intestine and the spleen. ". . . All of the tumors (hemangiomas) were considered to be simultaneous and independent formations—scattered over both lungs were numerous deep red, slightly elevated, round areas from 0.5 to 3 mm. in diameter . . . about fifty of these patches were counted on the surface of each lung."

It is fortunate that not all cases of pulmonary hemangioma are as diffuse as those just cited. The usual case of pulmonary arteriovenous fistula presents one or more cavernous hemangiomas in the lungs which are amenable to surgical excision.

There have been numerous excellent reviews of pulmonary arteriovenous fistula.[7,10,19,29,33,60,63,103,148] From a search of the world literature and from personal correspondence with the Diplomates of the American Board of Thoracic Surgery listed in the Directory of Medical Specialists (1951), we have collected 139 cases of pulmonary arteriovenous fistula. (Table 1.) We are reporting herein an additional case.

In addition, Haight[66] writes that he has had one case with bilateral lesions, and Kinsella[78] states that he has had seven cases of pulmonary arteriovenous fistula during the past year. Five of these cases occurred in one family and will be reported in detail later. This makes a total of 148 patients known to have pulmonary arteriovenous fistula.

CIRCULATORY DYNAMICS IN PULMONARY ARTERIOVENOUS FISTULA

In pulmonary arteriovenous fistula there is a shunt between the pulmonary artery and pulmonary vein. Unoxygenated blood flowing to the lungs is returned through the shunt, unoxygenated, to the heart. The effect upon the individual will depend upon the amount of unoxygenated blood so shunted back to the heart. When the shunt is small, no noticeable effect results. When a larger amount of unoxygenated blood is shunted back into the general circulation, the oxygen saturation of the blood falls and is manifested clinically by cyanosis and dyspnea. Clubbing of the fingers and toes develops. The chronic anoxia causes compensatory polycythemia to develop. In many of the reported cases the polycythemia is not evident, but rather a normal blood count or even anemia may be present. This is due to chronic hemorrhage from the lips, nose, mouth, lung or gastrointestinal tract, due to the hereditary hemorrhagic telangiectasis. The heart usually is not affected. The blood pressure is normal. Various central nervous system manifestations, varying from a sensation of tingling to convulsions and paralysis, are common due to the polycythemia and the anoxia.

In this connection it is interesting to note that any portion of the lung rendered atelectatic from any cause serves as a pulmonary arteriovenous fistula.

All of these pathologic effects are removed when the pulmonary arteriovenous fistula is removed.

DIFFERENTIAL DIAGNOSIS OF PULMONARY ARTERIOVENOUS FISTULA

It must be realized that pulmonary arteriovenous fistulas may, due to the small amount of blood shunted, produce no symptoms at all,[108] the only indication of its presence being a density in the lung seen on chest x-ray. It is probable that the hemangioma in the lung, being a part of the hereditary hemorrhagic telangiectasis, behaves in a similar manner to the telangiectases of the mucous membranes, that is, enlarges after puberty. It should be pointed out that although Wilkens gave a complete description of the disease in 1917, the clinical entity has only quite recently risen to the level of consciousness in the profession. Thus, "arteriovenous fistula of the lung usually masquerades in a cyanotic young patient with clubbing of fingers and toes as congenital heart disease or polycythemia until either a tumor of the lung or a continuous murmur in the chest is finally discovered." [76]

Probably the most important thing is to suspect the existence of a pulmonary arteriovenous fistula whenever a suspicious density is noted in the lung. The density is usually loculated and may present a grape-like cluster. Bands (the enlarged pulmonary artery and vein) may be seen connecting the density to the hilum.[52,113,121] Grishman et al.[65] state that the roentgenographic appearance of congenital arteriovenous aneurysm of the lung is rather characteristic: "a serpiginous racemose cluster, or coil of structures, with parallel walls which suggest vascular structures in density and outline." Increase in the intrathoracic pressure (Valsalva test) causes and vascular lesions to decrease in size, and decrease of the intrathoracic pressure (Müller test) causes the vascular lesions to increase in size.[121] Planograms are sometimes helpful. Angiocardiography, when properly performed, is usually diagnostic; however, several failures have been noted. One death occurred in this series[129] after angiocardiography with diodrast.

If one suspects the presence of a pulmonary arteriovenous fistula and listens over the area with a stethoscope, a murmur will be heard in most cases. This murmur is limited to a well circumscribed area which may be quite small and therefore easily missed. Also the presence of the small telangiectases of the lips, tongue, buccal and nasal membranes, with a history of bleeding therefrom, is indicative of heredi-

TABLE 1*

No.	Authors	Year	Age (yr), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
1	Churton	1897[26]	12, M, W	.....	+	.....	..	..	+
2	Wilkens	1917[143]	23, F, W	Since birth	+	+	+	..	+
3	DeLange and DeVries Robles	1923[31]	2½ mo., F, W	Since birth	..	.....	..	+	.....
4	Jaffe	1920[74]	4 days, F	Birth	..	About 100 hemangiomas of the skin plus large hemangioma of lt. side of neck	..	..	.....
5	Wollstein	1931[146]	4 mo , F, W	Birth	..	+	+	..	.....
6	Reading	1932[105]	4, F, N	Birth	..	0	+	+	+
7	Hall	1935[67]	40, F, W	.....	..	.....	..	..	.....
8	Bowers	1936[18]	2 days, M, W	2nd day	..	.....	..	..	.....
9	Rodes	1938[112]	25, M, W	Early life	+	+	+	+	0
10	Duvoir et al.	1939[37]	12, F, W	9	0	0	0	0	0
11	Smith and Horton	1939[127]	47, M, W	Blue baby	0	0	+	+	+
12	Plaut	1940[104]	35, F, W	Early life	..	.....	+	+	Over heart
13	Plaut	1940[104]	53, F, W	0	..	.....	..	..	.....
14	Hepburn and Dauphinee	1942[71]	23, F, W	15	0	.....	+	+	.....
15	Goldman	1943[61]	22, M, W	Since infancy	..	+	+	+	.....
16	Verse	1943[136]	3 days, M, W	Since birth	..	.....	..	..	.....
17	Verse	1943[136]	30, F, W	Too small to cause symptoms	..	.....	..	..	.....
18	Adams et al.	1944[3]	24, M, W	8	+	+	+	..	.....

* This chart contains all of the known cases of pulmonary arteriovenous fistula. (The one case of Haight and the seven of Kinsella are not included as details are not available.)

TABLE 1 (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
..	.....	.....	.....	0	Multiple	Autopsy July, 1895; no mention of cyanosis or clubbing
+	.....	.....	Multiple pulmonary aneurysms	0	L.L.L. R.M.L. R.L.L.	Beautiful clinical description with typical findings of pulmonary arteriovenous fistula; at autopsy the 3 pulmonary arteriovenous fistulas were carefully dissected
..	.....	.....	Meningitis	0	R.U.L. L.U.L.	Autopsy June 8, 1922; R.U.L. had 1 hemangioma 3 cm. in diameter; lt. lung 2 lesions 1.5 cm.
..	.....	.....	.....	0	About 50 small hemangiomas scattered over the surface of each lung	Autopsy; died at 9 mo. of age; hemangiomas of many organs; considered simultaneous; died of brain abscess
+	0	0	.....	0	R.U.L.	Autopsy December 4, 1930; large solid capillary hemangioma in R.U.L.; malignant form with generalized metastases
+	+	.....	Congenital heart	0	L.L.L.	Autopsy September 5, 1929; hemiplegia due to brain abscess
..	.....	.....	.....	.....	.....	Autopsy revealed 3,000 cc. bloody fluid in rt. chest cavity and hemorrhagic nodules throughout both lungs: "1. A case of malignant metastasizing hemangioma of which there are less than a dozen true cases reported. 2. The largest tumor was in the right lung. 3. True metastases consisting of both cavernous and malignant cellular areas were found in the lungs, nodes and liver."
..	.....	.....	.....	.....	R.L.L. L.L.L.	Large hemangioma in lt. lung had ruptured causing hemothorax; visceral hemangioma reviewed
+	+	0	.....	0	R.L.L. R.M.L. L.U.L.	Autopsy May, 1936; 1 hemangioma ruptured into bronchus causing death
+	0	0	Tuberculosis or tumor	11/10/33, exploration	L.L.L.	Autopsy 1938; died at 16 yr. of age from lagrippe; angioma of entire lower left lobe
+	+	+	Polycythemia rubra vera	0	R.L.L.	No autopsy; the first reported case in which diagnosis was made clinically
..	.....	.....	.....	0	All	Autopsy; showed multiple intravascular pulmonary hemangioendotheliomas and interventricular septum defect
..	.....	.....	.....	.....	One lobe	Autopsy; 1 small capillary angioma found in 1 lung; rheumatic heart with stenosis; angioma incidental
+	+	.....	Tuberculosis	2/14/40, rt. pneumonectomy	R.L.L.	Results good; first reported case successfully treated by operation; typical case; cavernous angioma R.L.L.
+	+	.....	Polycythemia rubra vera	11/1/45, lt. pneumonectomy	L.U.L.	Results good; brother to his second patient reported in 1947
..	.....	.....	.....	.....	Multiple, both lungs	Died January 3, 1928; autopsy; cavernous hemangioma, multiple, with rupture and massive lt. hemothorax
..	.....	.....	.....	.....	.....	Autopsy; very small capillary hemangioma found in 1 lung; incidental
+	+	.....	.....	(1) 2/43, lt. pneumonectomy; (2) 2/10/50, excision of fistulas rt. lung	L.U.L. R.U.L. R.M.L. R.L.L.	In 1949 multiple fistulas were demonstrated in rt. lung; operation rt. lung 2/10/50 (see Charbon, Adams and Carlson, 1952)

Abbreviations: R.U.L., right upper lobe; R.M.L., right middle lobe; R.L.L., right lower lobe; L.U.L., left upper lobe; L.L.L., left lower lobe.

TABLE 1 (*Continued*)

No.	Authors	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
19	Janes	1944[75]	30, M, W	29	+	+	+	..	+
20	Jones and Thompson	1944[76]	24, F, W	Since birth	..	.....	+	..	+
21	Alexander	1945[4]	41, M	For years	..	.....	+	+	.....
22	Rundles	1945[114]	56, M, W	14	+	+	..	..	+
23	Sisson et al.	1945[128]	45, F, N	22	+	.....	+	+	+
24	Lindgren	1946[84]	30, M, W	14	..	.....	+	..	+
25	Lindgren	1946[84]	29, M, W	Since childhood	+	+	+	+	+
26	Lindgren	1946[84]	25, F, W	11	+	.....	+	..	+
27	Makler and Zion	1946[89]	20, M, W	14	+	+	+	+	+
28	Beierwaltes and Byron	1947[13]	27, F, W	Since birth	+	+	+	+	.....
29	Bisgard	1947[14]	29, M	21	..	+	+	+	+
30	Burchell and Clagett	1947[21]	20, M	8	..	.....	+	+	+
31	Mallory et al.	1947[90]	23, F, W	12	+	.....	+	..	.....
32	Sweet	1947[130]	2, M	Since birth	..	.....	+	..	+
33	Salvesen	1947[115]	25, F, W	Since infancy	..	.....	+	..	+
34	Watson	1947[138]	27, M, W	0	0	0	0	0	.....
35	Watson	1947[138]	21, M, W	19	+	+	+	+	.....
36	Whitaker	1947[141]	44, F, W	Since birth	+	+	+	..	.....
37	Whitaker	1947[141]	33, M, W	.....	+	+	0	..	+
38	Williams and Flink	1947[144]	33, M, W	.....	+	+	+	+	+
39	Barnes et al.	1948[9]	17, F, W	3	+	.....	+	+	+

TABLE 1 (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	o	.....	.....	10/4/41, local excision R.L.L.; 6/25/42, local excision L.L.L.	R.L.L. L.L.L.	Results good; 2 hemangiomas were dissected from R.L.L. in 1941 and 2 from L.L.L. in 1942
+	+	.....	Tumor of lung, polycythemia vera	9/30/43, rt. pneumonectomy	R.U.L.	Results good; pneumothorax for 5 mo. ineffective
+	+	.....	Polycythemia vera	o	Multiple bilateral	Autopsy; coronary thrombosis and multiple pulmonary arteriovenous fistulas in both lungs
+	.....	.....	Pulmonary artery aneurysm	o	Rt. lung	Also had telangiectasis of gastric membranes and severe G.I. hemorrhage
+	Mild +	+	.....	o	R.M.L. L.L.L.	Died after attempted angiography; autopsy; died 25 min. after injection of 35 cc. 70 per cent diodrast; autopsy also revealed cavernous hemangioma of liver
+	o	.....	.....	o	L.L.L.	No autopsy; kyphosis since age of 12; increasing dyspnea on exertion
+	+	.....	.....	7/16/43, excision and dissection of 2 fistulas rt.; 3 small ones were left	R.U.L. R.L.L. Multiple bilateral	Family history positive for telangiectasis; severe C.N.S. symptoms; repeated hemoptyses; bilateral fistulas
+	.....	+	.....	5/25/45, lingulectomy	Lingula	Result good; numerous hospitalizations since age 11 for hemoptyses, weakness, cyanosis
+	+	.....	.....	o	R.M.L. L.U.L.	Chest x-ray in 1944 showed 3 masses in rt. lung and 1 in lt. lung
+	+	.....	.....	3/21/46, rt. lower lobectomy	R.L.L.	Results good
+	+	.....	.....	1946, rt. lower lobectomy	R.L.L.	Results good
+	+	.....	.....	Rt. middle lobectomy	R.M.L.	Results good; at operation much hemorrhage encountered due to collateral circulation from chest wall
+	+	.....	Tuberculosis, congenital heart disease	Rt. middle lobectomy	R.M.L.	Results good
+	+	.....	.....	Rt. middle lobectomy	R.M.L.	Results good
+	+	.....	.....	o	Rt. lung	Patient also had lymphogranulomatosis
+	o	.....	.....	2/28/46, ligation and division of artery	R.L.L.	Improved; no symptoms; lesion seen on routine x-ray on discharge from army
+	+	+	.....	8/8/46, rt. lower lobectomy	R.L.L.	Results good; had served 3 yr. with Marines prior to complaints; studied for 1 yr. in hospital; multiple phlebotomy
+	+	.....	.....	9/44, rt. lower lobectomy	R.L.L.	Died 6th p.o. day
+	.....	.....	.....	o	L.L.L.	Patient had no complaint except frequent nosebleed; refused operation
+	+	.....	Epilepsy	o	L.U.L.	Reported as hereditary hemorrhagic telangiectasis; Valsalva maneuver positive; planograms taken
+	+	.....	Congenital heart disease	9/12/46, lt. upper lobectomy	L.U.L.	Autopsy; died 3rd p.o. day of Staphylococcus tracheobronchitis

TABLE 1 (*Continued*)

No.	Authors	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
40	Barnes et al.	1948[9]	42, M, W	26	+	+	+	..	0
41	Boerema and Brilman	1948[17]	10, M, W	Since birth	+	+	+	+	.....
42	Brøbeck	1948[20]	34, F, W	Since infancy	..	+	+	+	+
43	Cleland	1948[27]	51, F, W	Since childhood	+	+	+	+	.....
44	Goldman	1948[63]	32, M, W	26	..	.....	+	..	+
45	Moyer and Ackermann	1948[97]	29, M, W	22	+	+	0	0	.....
46	Moyer and Ackerman	1948[97]	26, M, W	15	+	+	+	+	+
47	Moyer and Ackerman	1948[97]	28, M, W	23	+	+	+	..	.....
48	Maier et al.	1948[87]	20, F, W	Since infancy	+	.....	+	..	+
49	Packard and Waring	1948[101]	31, M, N	29	..	+	+	+	+
50	Samson	1948[117]	25, M, W	.....	..	.....	+	..	.....
51	Wodehouse	1948[145]	13, M, W	5	..	.....	+	+	.....
52	Wodehouse	1948[146]	21, M, W	0	..	.....	0	0	.....
53	Barker and Trounce	1949[8]	27, M, W	4	+	+	+	+	+
54	Barker and Trounce	1949[8]	13, M, W	5	..	.....	+	..	+
55	Beddard	1949[11]	36, M, W	.....	+	.....	0	..	+
56	Crane et al.	1949[29]	50, M, W	.....	..	.....	+	..	.....
57	Duisenberg and Arismendi	1949[38]	26, M, W	.....	+	.....	0	..	..
58	Erf et al.	1949[51]	20, M, W	4	+	.....	+	..	+
59	Ettinger et al.	1949[52]	34, F, W	24	+	.....	+	..	+

TABLE 1 (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	+	.....	Congenital heart disease	5/7/47, rt. lower and middle lobectomy	R.L.L. R.M.L.	Improved; patient a fine athlete; at 26 yr. of age noted to be cyanotic after hockey game; clubbing at 31 yr.; dyspnea and hemoptyses; developed empyema p.o.
+	+	.....	Tuberculosis (in sanatorium for 1 yr.), congenital heart disease	4/8/47, rt. upper and middle lobectomy	R.U.L. R.M.L.	Large telangiectasis back of neck; convulsions; p.o. able to play normally for first time
+	+	.....	.....	12/18/47, lingulectomy	R.U.L.	Results good; minor C.N.S. complaints; pain in both upper extremities, dizziness, headache
+	.....	+	Heart disease	7/25/47, rt. lower lobectomy	R.L.L. L.U.L. L.L.L.	Autopsy; died 20 hr. p.o.; liver showed surface hemangioma; case of generalized hemangiomatosis
+	+	.....	.....	0	L.U.L.	Brother to patient reported in 1943
+	0	+	.....	0	Lt. lung	Brother to second patient; whole family strongly positive for hereditary hemorrhagic telangiectases
+	+	+	.....	3/8/47, rt. pneumonectomy	R.L.L.	Results good; brother to first patient; the superficial telangiectases bled on exposure to sunlight
+	+	.....	.....	Lt. lower lobectomy	L.L.L.	Results good; the telangiectases of the head, nose and mouth still bleed
+	+	+	.....	11/20/46, rt. lower lobectomy	R.L.L.	Results good; patient had bacterial endarteritis in the fistula; also osteomyelitis of rib
+	+	.....	.....	12/8/44, ligation rt. pulmonary artery	R.U.L. R.L.L.	Results fair; patient Chinese; x-ray and examination 11/26/47 showed evidence of recanalization of ligated artery
+	+	.....	Unidentified infiltration R.U.L., asthma	4/47, rt. middle lobectomy	R.M.L.	Results good; lesion noted on routine chest x-ray; had "asthmatic history"
+	+	.....	Congenital heart disease	11/8/46, for brain abscess	R.L.L.	Autopsy; recurrent headaches; convulsions; hemophilus influenzae cultured from brain abscess
..	0	.....	.....	8/13/45, rt. lower lobectomy	R.L.L.	Patient served in Royal Canadian Air Force 2 yr.; no complaint; operated upon because of mass seen on chest x-ray
+	+	+	Congenital heart disease	4/5/48, rt. middle and lower lobectomy	R.L.L. L.L.L.	Died 30 hr. p.o.; small hemangioma found in L.L.L. at autopsy
+	+	+	Congenital heart disease	2/10/49, lt. upper lobectomy	L.U.L.	Results good
+	+	+	.....	Advised but refused	R.L.L.	Chief complaint, ache in rt. upper chest and rarely blood-streaked sputum
+	.....	+	.....	Rt. upper and middle lobectomy	R.M.L.	Routine chest x-ray on 11/24/46 revealed large tumor rt. lung
+	Mild	+	.....	8/9/47, rt. lower lobectomy	R.L.L.	Had 4 severe hemoptyses prior to admission; advise angiocardiography in every suspected case
+	+	+	Studied for 15 yr.	0	L.U.L.	Autopsy 5/11/47; death from left hemothorax (rupture of fistula); angiocardiograms unsuccessfu; also there was a large
+	+	.....	Tuberculosis	2/26/47, lt. lower lobectomy	L.L.L.	Results good; intermittent hemoptyses for 10 yr.; treated in sanatorium; pneumothorax for 2 yr.

TABLE 1 (*Continued*)

No.	Authors	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
60	Giampalmo and Giampalmo	1949[59]	21, M	Since infancy	+	.....	+	+	.....
61	Grishman et al.	1949[85]	23, M, W	.....	..	.....	+	..	.....
62	Grishman et al.	1949[85]	20, F, W	.....	..	.....	+	..	.....
63	Grishman et al.	1949[85]	.....	.....	+	.....	+	+	.....
64	Hedvall	1949[70]	51, F	0	0	0	0	0	.....
65	Yater et al.	1949[148]	42, F, W	10	..	.....	+	+	+
66	Yater et al.	1949[148]	42, M, W	.....	+	+	..	..	+
67	Zavodny	1949[149]	60, F, W	54	+	+	0	0	+
68	Baer et al.	1950[7]	21, M, W	13	+	.....	+	..	+
69	Carswell	1950[22]	20, M	18	0	0	0	0	.....
70	d'Allaines et al.	1950[30]	5, M, W	2	+	0	+	+	.....
71	d'Allaines et al.	1950[30]	18, F, W	5	+	0	+	0	0
72	Denolin et al.	1950[32]	19, M, W	15	0	0	+	..	+
73	Forsee et al.	1950[54]	20, M, W	.....	0	0	+	0	0
74	Friedlich et al.	1950[55]	M	5	..	.....	+	..	.....
75	Friedlich et al.	1950[55]	26, F	1½	..	.....	+	..	.....
76	Friedlich et al.	1950[55]	8, F	7	..	.....	+	..	.....
77	Lawrence and Rumel	1950[82]	30, F, W	24	..	0	0	..	+
78	Lawrence and Rumel	1950[82]	50, M, W	.....	..	0	+	..	+
79	Lequime et al.	1950[83]	65, F, W	20	+	+	+	..	.....

TABLE 1 (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	+	.....	.....	o	.....	.....
+	.....	+	.....	Rt. lower lobectomy	R.L.L.	.....
+	.....	+	.....	o	R.L.L.	.....
+	.....	.....	.....	Ligation of artery to fistula	R.L.L.	"At operation an accessory pulmonary artery was found entering the apex to the rt. lower lobe. Marked improvement followed ligation of vessel"
+	o	.....	Tuberculosis or metastatic tumor	o	R.U.L.	Noted on mass x-ray study; Valsalva's and Müller's tests positive; no symptoms; no change on x-ray over 7-year period
+	+	+	Congenital heart disease	2/13/48, rt. lower lobectomy	R.L.L.	Had had 6 pregnancies; had had "strokes" in 1940 and 1945; results good
+	o	+	.....	o	R.M.L.	Chief complaint, "pains in joints"; Osler-Weber's hereditary hemorrhagic telangiectasis with pulmonary angiomas
+	o	o	.....	o	R.L.L.	Anemia due to bleeding from telangiectases of G.I. tract; death from gastrointestinal hemorrhage; no autopsy
+	+	+	Polycythemia vera	1/6/48, rt. lower lobectomy; 5/12/48, lt. lower lobectomy	R.L.L. L.L.L. L.U.L.	Still cyanotic; on 9/28/48 lingulectomy was done for a large arteriovenous fistula in L.U.L.; had 3 operations; angiograms unsuccessful
+	Mild	.....	Tuberculoma	3/9/48, rt. lower lobectomy	R.L.L.	Chief complaint, interscapular pain; chest x-ray revealed a lesion; had mild clubbing of fingers
+	+	+	.....	2/16/50, lt. upper lobectomy	L.U.L.	Results good; chief complaint, cyanosis and dyspnea
+	+	+	.....	5/27/50, lt. lower lobectomy and lingulectomy	L.L.L. L.U.L.	Results good; chief complaint, dyspnea on exertion and coughing
+	o	o	.....	o	Lt. lung	Dyspnea, cyanosis and clubbed fingers; large pulsatile lesion lt. lung
+	+	o	Tuberculosis	10/21/48, lt. upper lobectomy	L.U.L.	Results good; lesion seen on x-ray when patient was discharged from army
o	+	.....	.....	Yes	.....	"At operation a large approximately thumb-sized communication was seen which arose from a branch of the left pulmonary artery and coursed into the left auricle. This was ligated and divided, following which there was dramatic improvement in the patient's color."
+	+	.....	.....	Rt. lower lobectomy	R.L.L.	Results good
+	+	.....	.....	Rt. lower lobectomy	R.L.L. R.M.L. R.U.L.	At operation smaller fistulas were noted in the other lobes
+	o	.....	.....	9/24/46, rt. lower lobectomy	R.L.L.	Results good; chief complaint, shortness of breath; no cyanosis or clubbing
+	+	+	.....	2/4/47, rt. upper and middle lobectomy	R.M.L.	Routine x-ray showed tumor of rt. lung; worked as laborer for years
+	+	.....	.....	o	Numerous bilateral	Disabled by severe dyspnea for 30 yr.; marked telangiectasis; autopsy 7/8/49

TABLE 1 (*Continued*)

No.	Authors	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
80	Lindskog et al.	1950[85]	24, M, W	.....	+	o	o	o	o
81	Lindskog et al.	1950[85]	56, F, W	Since infancy	+	.....	+	..	.....
82	Lindskog et al.	1950[85]	54, F, W	52	o	o	+	+	+
83	Lindskog et al.	1950[85]	55, F, W	.....	+	+	+	..	+
84	Santy et al.	1950[118]	46, M, W	40	o	o	+	+	o
85	Santy et al.	1950[118]	6, M, W	3 mo.	+	o	+	o	+
86	Segers et al.	1950[123]	63, F, W	48	o	o	o	o	o
87	Castellanos et al.	1950[23]	5, F, W	Since birth	..	.....	+	..	.....
88	Garland and Anning	1950[57]	6, M, W	3	+	+	+	..	+
89	Garland and Anning	1950[57]	8, F, W	7	+	+	+	..	+
90	Blades and Beattie	1951[16]	16, M, W	o	o	o	o	o	+
91	Blades and Beattie	1951[16]	Young adult, M	For 3 mo.	+	+	+	+	+
92	Blades and Beattie	1951[16]	20, M, W	6	..	o	+	+	+
93	Dotter and Steinberg	1951[34]	31, F, W	.....	..	.....	+	..	.....
94	Marchand et al.	1951[91]	24, M, W	Since childhood	+	+	+	..	+
95	Nightingale	1951[98]	18, M, W	o	o	o	o	o	+
96	Robb and Gottlieb	1951[107]	45, F, W	43	+	+	o	o	+
97	Roberts and Hutchison	1951[108]	66, F, W	.....	..	.....	o	..	+
98	Salvesen and Marstrander	1951[116]	37, M, W	21	..	+	+	..	.....

TABLE I (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	o	+	Tuberculosis	5/17/49, lt. upper lobectomy	L.U.L.	Results good; lesion diagnosed in 1941 but not operated upon until 1949 after patient had served 4 years in army
+	+	+	.....	1/3/50, rt. lower and middle lobectomy	R.L.L.	Results good; patient also had rheumatic heart disease; had had saddle embolus of aorta
+	+	+	.....	1/12/50, rt. lower and middle lobectomy	R.L.L.	Angiograms unsuccessful; had had radical mastectomy for ca.
+	o	o	.....	o	L.L.L. R.L.L.	See Kushlan, 1952; chronic osteomyelitis leg; had had brain abscesses; frequent profuse epistaxes
+	+	o	.....	2/4/49, lt. upper lobectomy	L.U.L.	Results good; chief complaint, effort dyspnea; profuse perspiration, cyanosis, headaches, bilateral deafness, vertigo
+	+	o	.....	11/19/49, lt. upper lobectomy	L.U.L.	Results good; chief complaint, cyanosis, hemoptysis, dyspnea
+	o	o	.....	o	Lt. lung	Chief complaint, effort dyspnea, blue lips; pulsatile shadow in left lung seen
+	.....	+	.....	Rt. lower lobectomy	R.L.L.	Results good; cyanosis noted since birth; no clubbing; angiocardiography on 2 occasions
..	+	.....	.....	Rt. middle lobectomy	R.M.L.	Results good; dilated vessels on surface of R.M.L.
+	+	.....	.....	Local excision	R.U.L.	Results good; at operation a hemangioma the size of a gallbladder was found lying in the fissure at the root of the lingula
+	+	+	.....	10/17/45, lt. lower lobectomy; 3/1/49, wedge resection, rt. lower lobectomy	L.L.L. R.L.L.	Results good; hospitalized because of "machinery murmur" heard over lt. posterior chest
+	Mild	+	.....	12/9/48, lt. lower lobectomy, lingulectomy; 3/17/49, rt. middle lobectomy	L.U.L. L.L.L. R.M.L. R.L.L.	Results good; still has 1 small fistula in R.L.L.
+	Mild	+	.....	12/1/49, rt. lower lobectomy	R.L.L.	Results good; shadow in R.L.L. seen on a routine chest x-ray
+	+	+	.....	o	L.L.L.	Refused surgery
+	+	.....	.....	12/50, rt. upper lobectomy, rt. middle lobectomy and rt. lower lobectomy	R.U.L. R.M.L. R.L.L.	Also had bacterial endocarditis
+	o	+	.....	o	R.M.L.	Lesion seen on mass x-ray study; no complaints
+	o	+	.....	8/22/50, lingulectomy	L.U.L. (lingula)	Results good; chief complaint, precordial pain, fatigue, shortness of breath
+	o	o	.....	o	R.L.L. R.U.L.	Lesion seen on routine chest x-ray; chief complaint, pain in back; 1 son had had an operation for arteriovenous aneurysm of the spinal canal
+	+	.....	Bronchitis	10/13/45, rt. middle lobectomy	R.M.L.	Died 5/16/48, 2½ yr. p.o. from subarachnoid hemorrhage

TABLE 1 (*Continued*)

No.	Authors	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
99	Salvesen and Marstrander	1951[116]	21, F, W	14	..	.....	+	+	.....
100	Salvesen and Marstrander	1951[116]	42, F, W	For years	..	.....	o	..	.....
101	Dubost et al.	1951[35]	29, M, W	7	..	+	+	..	+
102	Derra	1951[33]	27, F, W	8	+	+	+	+	.....
103	Szutrely and Erdelyi	1951[131]	5, F, W	2	+	+	+	..	+
104	Nixon and Perry	1952[99]	26, M, N	8	+	.....	+	..	+
105	Parker and Stallworth	1952[103]	33, F, N	33	+	o	+	+	+
106	Seaman and Goldman	1952[121]	79, F	.....	+	+	+	..	+
107	Seaman and Goldman	1952[121]	58, F	.....	+	+	o	..	+
108	Seaman and Goldman	1952[121]	55, F	.....	+	+	o	..	+
109	Seaman and Goldman	1952[121]	26, M	.....	+	+	+	..	+
110	Middehoven	1952[95]	6, F, W	Since infancy	..	.....	+	..	+
111	Andre et al.	1952[5]	46, F	23	o	o	+	..	+
112	Talbot and Silverman	1952[133]	48, M, W	o	+	o	o	+	o
113	Lodin	1952[88]	26, M, W	Since childhood	..	.....	+	+	+
114	Schludermann	1952[120]	44, F, W	44	+	+	o	o	+
115	Jones	1953[77]	37, M	.....	..	.....	+	..	.....
116	Jones	1953[77]	7, F	.....	..	.....	+	..	.....
117	Jones	1953[77]	29, F	.....	..	.....	+	..	.....
118	Jones	1953[77]	60, M	.....	..	.....	+	..	.....
119	Meckstroth	1953[94]	15, M, W	10	+	.....	+	+	.....
120	Miller	1953[98]	53, M	.....	..	.....	...	..	.....
121	Woods	1953[147]	35, F, W	.....	..	.....	..	..	+

TABLE I (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	+	.....	Congenital heart disease	o	Rt. lung	Died elsewhere May, 1935, after unrelated(?) operation
+	o	.....	Bronchitis	o	R.M.L.	Dyspneic for years; refused operation
..	+	+	.....	4/5/51, lt. lower lobectomy	L.L.L.	Transferred to medical service; very large fistula; had p.o. hemorrhage and embolism
+	+	+	.....	Rt. lower lobectomy	R.L.L.	Results good; severe C.N.S. symptoms; marked cyanosis since 8 yr. of age
+	+	+	.....	o	Multiple bilateral	Autopsy 3/15/50; died of massive G.I. hemorrhage; also had heart failure
+	+	.....	Congenital heart disease	1/28/50, lt. pneumonectomy	L.L.L.	Results good; chief complaint, pain in neck; effort dyspnea; known as "blue boy"; also has hemangioma of 3rd cervical vertebra
+	o	+	.....	9/16/49, excision of fistula	.....	Not cured; chief complaint, substernal pain; the presence of another fistula in the rt. lung is suspected
+	+	.....	.....	.....	.....	Cases to be reported in detail elsewhere
+	o	.....	.....	.....	.....	Cases to be reported in detail elsewhere
+	o	.....	.....	.....	.....	Cases to be reported in detail elsewhere
+	+	.....	.....	.....	.....	Cases to be reported in detail elsewhere
+	+	.....	.....	6/18/52, rt. upper lobectomy	R.U.L.	Results good; blue child; could never play actively; marked clubbing of fingers and toes which disappeared p.o.
+	+	+	Heart disease, polycythemia	3/24/52, lt. lower lobectomy	L.L.L.	Results good; Valsalva test positive; tomographs helpful; bronchospirography diagnostic
+	o	o	.....	5/22/50, lingulectomy	L.U.L. (lingula)	Results good; picked up on routine chest x-ray; rare nosebleed; asymptomatic
+	+	.....	Congenital heart disease	.....	Multiple R.L.L.	Abscess of brain and meningitis developed; autopsy also revealed arteriovenous fistulas in R.L.L.
+	Anemic	Anemic	.....	o	R.L.L.	Severe gastrointestinal hemorrhage; telangiectasis
+	+	.....	.....	12/20/49, rt. pneumonectomy	R.M.L.	Rt. pneumonectomy because of active tuberculosis rt. lung; previously treated by pneumothorax
+	+	.....	.....	o	Lt. mid-lung field	Died from rupture of aneurysm
+	+	.....	.....	o	Rt. mid-lung	Advised to have surgery
+	+	.....	.....	o	R.M.L.	Has hypertension and myocardial disease
+	+	+	.....	4/49, rt. lower lobectomy; 1/30/53, 4 fistulas ligated, left	R.L.L., multiple bilateral	At operation 1/30/53 about 50 aneurysms were seen; patient now receiving x-ray treatments to rt. chest
+	o	.....	Cancer of rt. lung, tuberculoma	7/3/50, dissection and excision of fistula	R.U.L.	On 5/4/51 he had a "stroke" with unconsciousness and convulsions; died 5/15/51; autopsy showed rupture of cerebral aneurysm
+	+	.....	.....	Lt. upper lobectomy	Lingula R.M.L.	Slightly improved; patient also had rheumatic heart disease and mitral stenosis; cousin to Case 122; small bilateral fistulas

TABLE 1 (*Continued*)

No	Author	Year	Age (yr.), Sex and Race	Age at Onset of Symptoms (yr.)	Epistaxis or Hemoptyses	Telangiectasis	Cyanosis, Clubbing, Dyspnea	C. N. S. Symptoms	Murmur over Fistula
122	Woods	1953[147]	45, F, W	.....	..	.....	..	..	.....
123	Woods	1953[147]	Young, F, W	.....	+	.....	..	..	+
124	Higginson	1953[72]	56, M	.....	..	+	..	..	+
125	Higginson and Goodman	1953[72]	M, W	Since infancy	+	+	+	+	+
126	Tuttle and O'Rourke	1953[135]	F, W	.....	..	.....	..	+	.....
127	Laird	1953[81]	31, M, W	27	+	+	+	..	+
128	Schaffner	1953[119]	23, M, W	0	..	.....	0	..	+
129	Shaw	1953[124]	20, F, W	.....	..	.....	+	..	.....
130	Conklin	1953[28]	30, M, W	Since childhood	..	+	+	..	+
131	Conklin	1953[28]	35, M, W	.....	..	+	+	..	+
132	Conklin	1953[28]	70, F, W	25	+	.....	..	..	+
133	McCormick	1953[93]	63, F, W	6 mo.	0	.....	..	..	0
134	Robertson	1953[110]	24, M, Indian	22	..	.....	+	..	+
135	Weisel	1953[140]	58, M, W	Since childhood	..	+	..	..	+
136	Mayer	1953[92]	45(?), F, W	0	..	.....	0	..	.....
137	Tobin and Wilder	1953[134]	46, M, W	Since childhood	+	+	+	..	+
138	Tobin and Wilder	1953[134]	17, M, W	Since childhood	+	+	0	..	+
139	Israel and Garfield	1953[73]	41, F, W	18	+	+	0	..	0
140	Stringer	1953	26, M, W	Since childhood	+	+	+	+	+

TABLE I (*Continued*)

Lesion on Chest X-ray	Polycythemia	Angiocardiography	Mistaken Diagnosis	Operation	Lobes Involved	Remarks
+	.....	.....	.....	Rt. middle lobectomy	Small bilateral	Died 48 hr. p.o. from cerebral embolism originating at the site of the removed pulmonary fistula; cousin to Case 121; autopsy
+	+	.....	.....	Rt. lower lobectomy	Large R.L.L.	Seven mo. pregnant when seen; chief complaint, severe pulmonary hemorrhage; died suddenly 1 yr. p.o.; no autopsy
+	o	+	.....	Lt. lower lobectomy	L.L.L.	Results good; coin lesion in lt. chest
+	+	+	.....	1/53, rt. middle lobectomy, rt. lower lobectomy	R.L.L. R.M.L. Lingula L.L.L.	Patient and his father had been diagnosed as having congenital telangiectasis; operation on lt. lung planned in future
+	.....	.....	Epilepsy, grand mal	5/26/52, left lower lobectomy	L.L.L.	Patient also diagnosed as having tuberculosis
+	+	.....	Tuberculosis	2/14/53, lt. lower lobectomy	L.L.L.	Results good; chief complaint, recurrent stiff neck; recurrent epistaxis and peculiar sensation in chest
+	+	.....	.....	Plans to have surgery	R.L.L.	College student had had yearly chest x-rays since 1947; no symptoms; lesion first appeared in 1951
+	.....	.....	.....	1952, posterior segment L.U.L. removed	L.U.L.	Results good; case to be reported in detail elsewhere
+	+	+	.....	5/6/52, superior segment R.L.L.	R.L.L.	Results good; "The large fistula presented itself under the visceral pleura and was removed with little lung tissue"
+	.....	+	.....	2/15/52, basal segment R.L.L.; suture small fistula R.M.L.	R.M.L. R.L.L.	Results good; lesion in lung picked up on routine chest x-ray
+	.....	+	.....	o	R.L.L.	Had a cardiovascular accident November, 1952; not followed since
+	+	.....	Cancer of rt. middle lobe	10/16/51, rt. pneumonectomy	R.M.L.	Patient died on operating table due to attenuation of the rt. pulmonary artery; it tore and patient died from hemorrhage
+	+	.....	.....	6/7/45, rt. lower lobectomy; not relieved; 9/18/46, rt. pneumonectomy attempted	R.M.L. R.L.L.	Died at operation; received shrapnel wound rt. lung 8/13/44, following which polycythemia and signs of rt. pulmonary arteriovenous fistula developed
..	Anemic	+	.....	12/2/52, rt. lower lobectomy	R.L.L.	Results good; multiple telangiectasis of face, nose, mouth and rectum
..	.....	.....	Not made prior to autopsy	o	Multiple, rt. lung	Patient died of malignant brain tumor; 2 arteriovenous fistulas were found in the rt. lung at autopsy
+	o	o	.....	Lt. lower lobectomy	L.L.L.	Results good; father to case below; severe nasal hemorrhages
+	o	.....	.....	Advised	L.L.L.	Planography positive; Valsalva and Müller tests positive
+	o	.....	.....	Advised	L.L.L.	Surgery was advised in April, 1949; patient hemorrhaged from the ruptured fistula April, 1950, and died; autopsy; angiomas also in liver and spleen
+	o	o	.....	11/6/52, rt. middle lobectomy	R.M.L.	Results good; patient had been troubled with frequent nosebleeds and headaches all his life

tary hemorrhagic telangiectasis and should alert one to look for pulmonary hemangioma. Cyanosis, clubbing of the fingers and toes, and dyspnea are also typically present, but depend upon the quantity of unoxygenated blood shunted into the pulmonary vein. Hemorrhage from the pulmonary hemangioma may occur intrapleurally and/or intrabronchially and be fatal.

Other diseases commonly confused with pulmonary arteriovenous fistula are as follows:

Polycythemia Rubra Vera. In those cases of pulmonary arteriovenous fistula in which polycythemia is present, polycythemia rubra vera may be diagnosed. In the latter condition multiple, evanescent, spheric homogeneous densities may appear in the lung fields to confuse the picture. In polycythemia rubra vera the blood and bone marrow show immature white blood cells and basophilic stippling of the red cells.

Congenital Heart Disease. The presence of cyanosis, clubbing of the digits and dyspnea commonly results in confusion of pulmonary arteriovenous fistula with congenital heart disease. In congenital heart disease the presence of cardiac murmurs and the abnormal cardiac outline should be helpful in establishing the diagnosis. The heart is usually not affected by the presence of a pulmonary arteriovenous fistula.

Tuberculosis. Several cases of pulmonary arteriovenous fistula have been misdiagnosed as tuberculosis and the patients have been treated in sanatoriums. Thus Forsee et al.[54] reported a case in which: "On two occasions his sputum was reported as positive on smear examination for acid fast bacilli, and the infiltration was considered as tuberculous." Later the true nature of the lesion was suspected and the involved lobe removed. The pathologist reported that there was no evidence of tuberculosis.

Benign and Malignant Pulmonary Tumors. Benign and malignant tumors may at first be suspected when the density due to the pulmonary arteriovenous fistula is seen on the chest x-ray. However, if one thinks of pulmonary arteriovenous fistula, the diagnosis will be greatly facilitated.

TREATMENT OF PULMONARY ARTERIOVENOUS FISTULA

The first patient with pulmonary arteriovenous fistula cured by operation was reported by Hepburn and Dauphinee in 1942.[71,125] In 1932 at the age of fifteen years the patient had cyanosis and dyspnea, and was diagnosed as having a "leaky valve." In 1938, because of symptoms referable to the lungs, she was sent to a tuberculosis sanatorium where cyanosis and clubbing of the fingers and toes was noted. A chest x-ray revealed a lesion in the right middle lobe. The blood count was normal, i. e., red blood cells 4,700,000, hemoglobin 14.8 gm. (95 per cent). She was seen in January, 1940, at the age of twenty-three years with the chief complaint of dizziness, faintness and thick speech. Cyanosis, clubbing of the fingers and toes, and dyspnea were noted. Marked polycythemia was present; the red blood count was 9,600,000 and hemoglobin 22.8 gm. (146 per cent). Artificial pneumothorax, right lung, was instituted with good collapse but no improvement. On February 14, 1940, right pneumonectomy was performed by Shenstone and Janes. The right lower lobe was found to contain a "cavernous angioma." In April, 1941, the patient was asymptomatic; the clubbing of the fingers and toes had receded. The patient was pregnant and was doing well.

As the nature of pulmonary arteriovenous fistula became evident and the possibility and frequency of multiple lesions in one or both lungs began to be appreciated, the desirability of leaving as much lung in the patient as possible became evident. Illustrative of a patient presenting multiple pulmonary arteriovenous fistulas is the patient first presented in 1944 by Adams et al.[3] and again in 1952 by Charbon et al.[25] He was seen on January 24, 1943, at the age of twenty-four, with the chief complaint of frequent nosebleeds and colds. Cyanosis, clubbing of the fingers and toes, hemangiomas of the lips, marked polycythemia, a small opacity in the right lung field (on chest x-ray) and a large density in the left lung field were noted. Left pneumonectomy was performed on February 11, 1943. The pathologic diagnosis was "multiple arteriovenous fistulas of the left lung." The patient improved. However, a bronchopleural fistula developed with empyema, which was diagnosed in 1945 and operated upon (left thoracoplasty) in 1948. Mild polycythemia was noted. In 1949 and 1950 cyanosis, clubbing of the digits and polycythemia had increased. Pulmonary angiograms revealed four cavernous spaces in the lower half of the right chest. After due consideration and special preparations the remain-

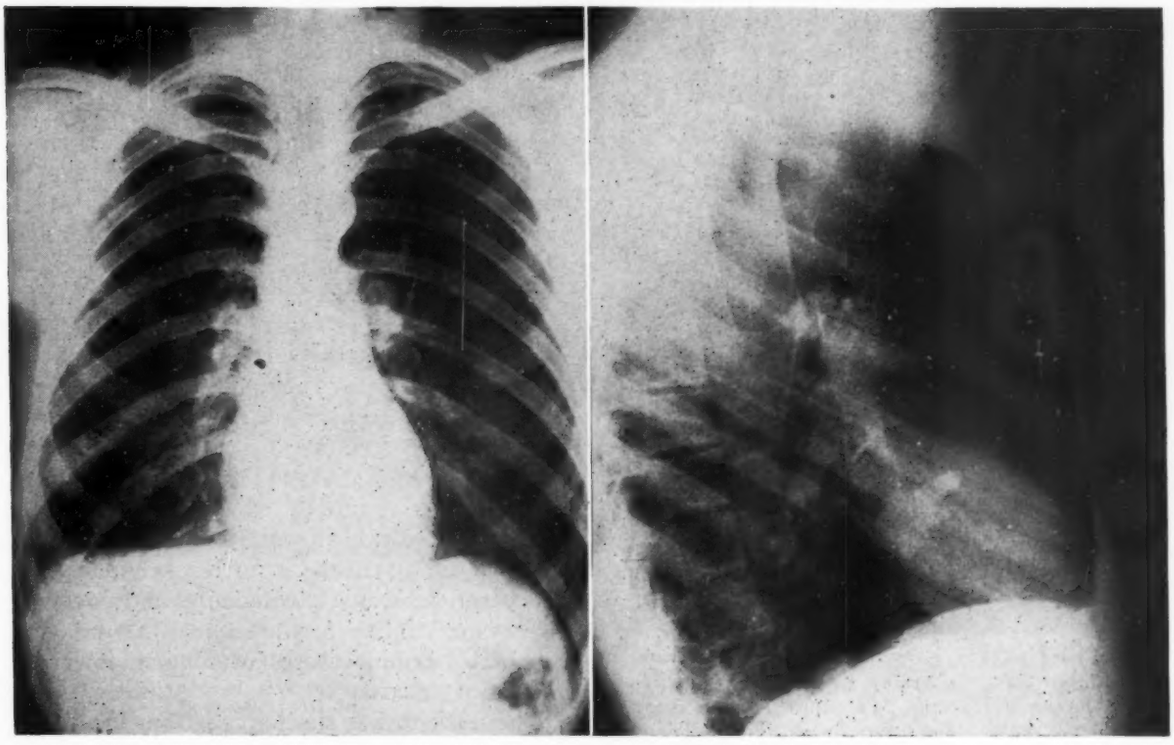

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FIG. 1. Case No. 5273. Posterio-anterior film. Two circumscribed densities in the right lower lateral lung field. These densities overlie the posterolateral course of the ninth rib.

FIG. 2. Same case. Lateral film. Multiloculated density in the lower central part of the right lung field. Directly over this density in the right axilla over a sharply circumscribed area a loud continuous murmur with a systolic accentuation could be heard. No other murmurs could be heard over other parts of the lung or heart.

ing (right) lung was operated upon February 10, 1950.[25] One interested in operating upon such a patient with multiple pulmonary arteriovenous fistulas in one lung with the other lung having been removed previously for the same disease should read this paper.[25] "Operation. The right thorax was entered through the bed of the seventh rib, which was removed subperiosteally. The entire pleural space was found to be obliterated. Adhesions were freed over the right middle and lower lobes. One arteriovenous fistula was found at the surface of the lung at the lower medial aspect of the right lower lobe, and another was found at the lower lateral aspect of the right middle lobe. The fistulas were noted to pulsate, but no thrill was palpated. The two areas containing fistulas were clamped off with right angle clamps and resected. At about the time of closure of the second fistula, the blood pressure dropped to 50/30 mm. Hg. Transfusion with plasma failed to produce a satisfactory rise in system pressure. Since the right ventricular pressure was only 30 mm. Hg. (well within normal limits), it was decided to give whole blood. The systemic blood pressure rose to 80/40 soon after this transfusion. Freeing of the adhesions over the right upper lobe was next begun. As the posterolateral aspect of this lobe was approached, a thrill could be palpated. A third arteriovenous fistula, located in the upper lobe, was broken into and bled considerably, causing a blood loss of 300 to 400 cc. Clamps were placed proximal to control the bleeding. This fistula was then obliterated with multiple interrupted sutures. There appeared to be a fourth fistula deep in the substance of the lower lobe, but it was felt not justified to attempt its removal. Two Pezzar catheters were brought through the eighth interspace, and the wound was then closed in layers. The patient's condition remained relatively good throughout the procedure. At the present time (January 1951) the patient is in fairly good condition. At rest he is not cyanotic, and is able to do some light exercise."

FIG. 3. Case No. 5273. X-ray film of the resected right middle lobe with the artery having been injected with radiopaque material. The four hemostats on the right are on the hilar junctures, while the hemostat on the left points to the arteriovenous fistula. The enlarged artery (above the vein) can be seen arching over to the fistula, and the enlarged vein (below the artery) can be seen passing back to the hilum. Following this the specimen was dissected, and the cavernous nature of the arteriovenous fistula was demonstrated.

Multiple operations for multiple pulmonary arteriovenous fistulas have also been reported by Behrend and Baer.[12,72,75,94,110] Lobectomy is frequently mandatory due to the location of the lesion but, whenever possible, simple excision of the fistula,[16,20,28,72,75,84,96,103,124,133] is the procedure of choice. Other conditions or accidents, other than multiplicity of the pulmonary arteriovenous fistula, which have been encountered at operation are: injury to bronchus requiring removal of an additional lobe;[8] enlarged collateral circulation from the chest wall;[21] communication between the aorta and pulmonary artery;[51] communication between the pulmonary artery and the left auricle;[55] accessory pulmonary artery;[65] absence of the right middle lobe;[76] fatal hemorrhage from greatly dilated vessels to the fistula;[93] dense adhesions, the lysis of which resulted in shock and death.[110]

CASE REPORT

The patient (No. 5273), a twenty-six year old white man, was admitted to the hospital on November 3, 1952, with the diagnosis of pulmonary arteriovenous fistula of the right middle lobe of the lung. The patient's chief complaints were: headaches, nosebleeds, shortness of breath and tiring easily on exertion.

The patient stated that he had had frequent nosebleeds all his life; also, he had had severe headaches as far back as he could remember. He had "blacked out" several times and, according to his mother, had had "spells" when a child. His fingers and toes had been clubbed as far back as he could recall. He was inducted into the army in February, 1951, but was unable to keep up with the other men because of headaches, frequent nosebleeds and fatigability. His headaches were characterized by blurred vision and blindness preceding the headache. He was hospitalized from March 6 to April 17, 1951, at Camp Rucker, Alabama. The diagnosis was: (1) congenital arteriovenous aneurysm, right middle lobe; (2) multiple congenital telangiectases of tongue, lower lip and nasal mucosa.

The patient was discharged from the army. On July 28, 1952, he came under the care of one of us (R. C. B.), and the diagnosis of hereditary hemorrhagic telangiectasis with pulmonary arteriovenous fistula of the right middle lobe was confirmed. At this time a blood count showed marked anemia (hemoglobin 6.65 gm., red blood count 3,800,000). Electrocardiograms and a sound tracing of the murmur heard in the right axilla were secured. Ethinyl estradiol therapy, 0.5 mg. daily, was started on August 27, 1952. Following this there was a marked decrease in the number and frequency of the nosebleeds.

The patient stated that he had had measles, mumps, whooping cough and chicken pox. He had had pneumonia and pleurisy on the right side of the chest before he entered the army in 1951, and also again about two months before the present admission. He was sick in bed for one week at that time. There was no history of gastrointestinal bleeding or hematuria, and no bleeding other than that from the nose. He had undergone left inguinal hernia repair when twenty years of age. Married, he had no children.

Physical examination revealed the following: The patient was 5 feet, 10 inches in height and weighed 125 pounds. His temperature was 98.6°F., pulse 70/min., respirations 20/min., and blood pressure 110/60 mm. Hg. The

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FIG. 4. Case No. 5273. Photograph of the patient's hands four months after resection of the pulmonary arteriovenous fistula shows only minimal clubbing to be present. Prior to operation marked clubbing had been present.

FIG. 5. Same case. Photograph of the patient's toes four months after resection of the pulmonary arteriovenous fistula shows only minimal clubbing to be present. Prior to operation marked clubbing of the toes had been present.

patient was a thin, emaciated-appearing white man, slightly cyanotic. In the mucous membranes of the mouth, tongue, lips and nose there were numerous small, raised, red-colored papules. The mucous membranes were cyanotic. The head, ears, eyes, nose and throat, otherwise showed no abnormality. The heart was regular in rhythm with no murmurs or enlargement. The lungs were clear with no rales. In the right mid-axillary line at the level of the nipple there was a 5 cm. area where a loud to and fro murmur was heard. The murmur could be heard only in this well circumscribed area. There was marked predominance of the systolic component of the murmur. The abdomen was soft with no masses or tenderness. The spleen and liver were not palpable. An old left inguinal herniorrhaphy scar was present. The genitalia were normal. Rectal examination was negative. There was clubbing of all fingers and toes.

Laboratory reports were as follows: *November 4, 1952, urinalysis:* specific gravity, 1.027; reaction, acid; color, hazy amber; sugar, albumin and blood, negative; no microscopic findings.

November 4th, blood count: red blood cells, 4,400,000; hemoglobin, 13.5 gm. (81 per cent); white blood cells, 6,250. Differential: polymorphonuclears, 64 per cent; lymphocytes, 34 per cent; monocytes, 2 per cent.

November 6th, blood count: red blood cells, 5,600,000; hemoglobin, 12.5 gm. (80.6 per cent); white blood cells, 14,050. Differential: polymorphonuclears, 38 per cent; lymphocytes, 53 per cent; monocytes, 1 per cent; eosinophils, 8 per cent; P.C.V., 43 cc.; M.C.V. 81 c.μ.

Chest x-rays show loculated densities in the lower lateral aspect of the right lung field. (Figs. 1 and 2.)

Operation was performed on November 6th using oxygen, nitrous oxide and ether anesthesia with an endotracheal tube. The patient was placed in a lateral position. Through a right posterolateral incision the right fifth rib was resected. On exploration of the chest dense adhesions bound the upper and middle lobes to the lateral chest wall; also the fissure between the upper and middle lobes had been obliterated. Considerable dissection was necessary to free the lobes. The arteriovenous fistula was noted to be subpleural in the right middle lobe. No other lesions were noted. Right middle lobectomy was performed. Marked enlargement of the artery and vein to this lobe was noted. The pleural space was washed out, the remaining lobes were re-expanded and, after a thoracotomy drainage tube had been placed, the chest wall was closed in layers using interrupted silk and cotton sutures.

The resected specimen, i. e., the right middle lobe, was inflated and lipiodol was injected into

the large artery. X-rays were then taken showing the enlarged artery leading into the aneurysm and the enlarged vein draining it. (Fig. 3.) The specimen was then dissected. Microscopic studies revealed no other pathologic condition.

The postoperative course was uneventful. The skin sutures were removed on the seventh postoperative day. The patient was discharged on November 16th.

Follow-up examinations reveal that the patient is feeling better than he ever did. He is now able to do regular work in an automobile factory. The clubbing of the digits has almost completely subsided. The telangiectases of the oral membranes are not prominent. Four months postoperatively the clubbing of the fingers and toes had greatly decreased. (Figs. 4 and 5.)

Comment. This patient presents the typical history and findings characteristic of pulmonary arteriovenous fistula. The frequent and severe nosebleeds alleviated the polycythemia, even produced anemia. Operation removed the fistula with excellent results.

SUMMARY

From the world literature as well as from personal correspondence we have collected 147 cases of pulmonary arteriovenous fistula and have added one case of our own, making 148 known cases. Pulmonary arteriovenous fistula is a variant of hereditary hemorrhagic telangiectasis. The lesions in the lung tend to be multiple. The pathologic physiology of pulmonary arteriovenous fistula is due to the shunt between the pulmonary artery and vein. Unoxygenated blood is thereby shunted back to the heart and to the systemic circulation. The degree of symptoms depends upon the amount of unoxygenated blood shunted from the pulmonary artery into the pulmonary vein. When the shunt is small, no symptoms result. In a typical case of pulmonary arteriovenous fistula the characteristic findings are telangiectasis, cyanosis, clubbing of the fingers and toes, exertional dyspnea, polycythemia (frequently alleviated by hemorrhage from the telangiectases), an opacity (or opacities) seen in the chest x-ray film and a murmur heard over the lesion in the lung. Central nervous system manifestations ranging from attacks of numbness, tingling, headaches, dizziness, transient weakness and paralysis to loss of consciousness, paralysis, convulsions and brain abscess are of frequent occurrence. Treatment is surgical excision of the fistula. Lung tissue should be conserved as pulmonary arteriovenous fistula tends to be multiple and may occur in both lungs necessitating bilateral thoracotomy.

Additional Case. This thirty-four year old woman was admitted to the psychopathic hospital on June 5, 1954, suffering from catatonic stuperous episodes alternating with a paranoid state of one month's duration. Answers to questions were usually irrelevant. Throughout her lifetime this patient had shown cyanosis of the skin and mucous membrane, and the fingers and toes were clubbed. X-ray of her chest on admission had shown "a tumor" in the right lung, and it was the opinion of the psychiatrist that there might be some relationship between the acute mental condition and the chest findings. When seen on June 8th she was in coma, and a soft to and fro murmur was heard over the right middle lobe in an area about 3 cm. in diameter. Oxygen therapy improved her condition somewhat. The right middle lobe containing an arteriovenous fistula was resected on June 22nd. Her color immediately became normal following operation. Also, her condition changed from one resembling catatonia to one of extreme overactivity bordering on violence, during which time she was always acutely aware of her environment. Although she had been refusing food and had to be tube-fed, she soon began eating huge quantities of food and there was a great improvement in both her physical and mental condition. She was returned to her home on August 15, 1954, and her discharge made permanent on September 14th. She has remained in good mental condition since that time.

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Hidradenoma of the Anus

IRVING ROSENBERG, M.D. AND PHILIP V. SKERRETT, M.D., *Philadelphia, Pennsylvania*

From the Proctologic Service, Einstein Center, Eastern Division, and the Department of Laboratories, Einstein Center, Northern Division, Philadelphia, Pa.

HIDRADENOMA is an uncommon tumor. It may arise in any of the sweat glands, but usually in those of apocrine type. Most of the reported incidences have been located on the vulva, usually the labium majus.

In 1904 Pick described two vulval tumors of sweat gland origin and gave them the name by which they have since been designated. Approximately 200 cases[3–5,7,8,10,11,13] of hidradenoma have since been reported. The number in regions other than the vulva are few. In 1933 Ullman reported a hidradenoma in the breast of a thirty-five year old woman. In the same year Sezary and Levy-Coblentz[11] reported one from the forehead of a forty-four year old woman. In 1944 Cooper and McDonald[2] published an article on the first such tumor from the anus. In 1954, Teloh[12] reported on four cases from the anus collected in the previous fifteen years. All were in women. The first resembled a sinus tract with a painless perianal swelling. The second was a fleshy tumor at the mucocutaneous margin of the anus. The third was in a woman who complained of a small swelling at the anal margin; it resembled a fibrous tumor. The fourth was a polypoid perianal growth.

We are reporting two additional cases, representing the sixth and seventh cases of hidradenoma of the anus.

CASE REPORTS

CASE I. Mrs. A. F., age fifty-five, was seen on March 30, 1953, complaining of rectal discomfort. The anus appeared normal on digital and proctoscopic examination. On the left anterior aspect of the anus at the mucocutaneous junction was a lesion, ¼ inch in diameter, surrounded by a pearly hard collar and protruding through a beefy red mass which did not bleed on touch. It was not painful. Sigmoidoscopy was performed and was completely negative for a distance of 10 inches. The lesion was thought to be either a granuloma or an early malignancy. Biopsy was advised.

On April 2, 1953, under local anesthesia the tumor was entirely removed by one of us (I. R.). This was accomplished in the office without incident. Suturing was not necessary and healing progressed normally. One year later the area was completely healed and without evidence of local recurrence.

The pathologic report revealed the following: The specimen was an ellipse of anal skin, 1.5 by 0.8 cm. Centrally there was a pink, exophitic growth, 4 mm. in diameter, with a rough granular surface. It was surrounded by uninvolved skin. On microscopic examination, corresponding to the exophitic growth, a small papillary tumor protruded above the general epidermal surface. (Fig. 1.) A papillary adenomatous tumor lay partly beneath the level of the epidermis and extended through what probably was the duct of the gland of origin. The tumor was formed by columnar epithelial cells arranged along delicate fibrous stalks. (Fig. 2.) The individual tumor cells were quite uniform. There was no true capsule, but surrounding the tumor there was a condensation of collagenous fibers. The surrounding stroma showed mild mononuclear infiltration. At either side of the tumor the anal skin was covered by keratinizing squamous epithelium. Beneath there was some edema and dilatation of lymphatics. The diagnosis was: hidradenoma of perianal skin.

CASE II.* Patient E. R., age thirty-four, had a small pea-sized mass to the left of the anus and about ½ inch from the mucocutane-

* Contributed through the courtesy of Dr. William Antopol, Beth Israel Hospital, New York, N. Y.

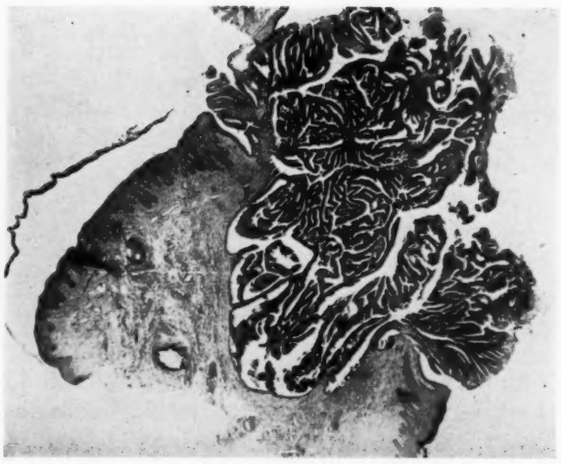

FIG. 1. Case 1. (1103-53 A.E.M.C., N.D.) Illustration shows papillary partly encysted tumor extending through the epidermal surface.

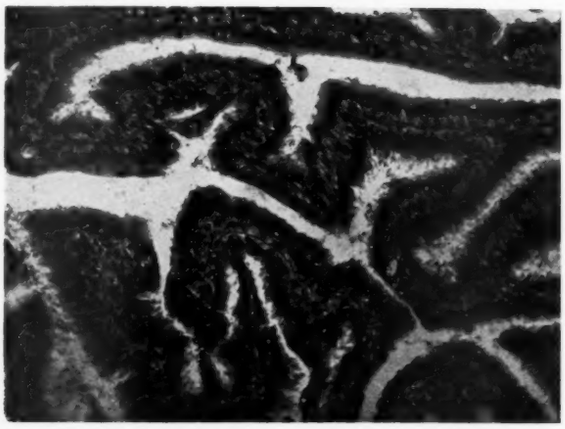

FIG. 2. Case 1. Higher magnification, showing columnar epithelial cells (with tendency toward stratification) arranged on delicate fibrous stalks.

ous line. The tumor was elevated above the skin surface in practically its entire diameter. It was completely excised.

Pathologic report revealed the following: The specimen was a roughly spherical nodule, 7 mm. in diameter. About three-quarters of the surface was covered with a thin, shiny skin-like tissue. The cut surface was semi-fluid and yellow-grey. On microscopic examination an intracystic papillary tumor was found beneath the intact epidermis. The papillae showed columnar epithelium covering a delicate fibrous stroma. In places the epithelium was stratified, two to three cells in thickness. Occasional macrophages with foamy cytoplasm were found both in the stroma and in the epithelium-lined spaces. The tumor had no true capsule, but was confined by the surrounding compressed connective tissue. The diagnosis was hidradenoma of anus.

A search of the standard proctologic texts does not reveal mention of this tumor. Brief mention of hidradenoma of the anus is found in textbooks of surgical pathology.[1,6] The gynecologists have described the gross characteristics. The tumor is generally firm, elevated and does not bleed on touch. Its color has been described as being either pale pink, red, grey or blue. Pain has not been noted. Its size usually does not exceed 5 cm. It may be confused with sebaceous cyst, other benign tumors or carcinoma.

Microscopically these tumors show a very characteristic picture. As originally described by Pick,[9] they arise from the epithelium of sweat glands. Most recent authors agree that they usually arise from the apocrine type of sweat gland. This probably accounts for the anatomic distribution of these tumors, for apocrine glands are most frequent in the axilla, about the nipples of the breast and in the genital skin. The hidradenoma is formed with columnar epithelial cells arranged on delicate fibrous stalks. There is a tendency towards stratification, as is also found in the apocrine gland. The tumors frequently have an intracystic papillary structure, resembling somewhat the intracystic papillary tumors of the breast. The great majority of hidradenomas are benign, although hidradenoid carcinoma has been described.

It is probable that hidradenoma of the anus is of greater frequency than the few reported cases would indicate. It is important to differentiate these tumors from the other tumors which occur in the region of the anus. It is advisable that all anal and perianal tumors be excised and subjected to histologic examination.

SUMMARY

Two cases of hidradenoma of the anus are reported. This brings the total number of reported cases to seven. These tumors are adenomas of the apocrine sweat glands and are usually benign. Characteristically they are well encapsulated, intracystic papillary tumors. They are lined by a single layer of columnar epithelium which has a tendency towards stratification.

These tumors raise a diagnostic problem for the proctologist. The gross appearance of the

lesion may well be mistaken for carcinoma. They should be included in the differential diagnosis of anal tumors.

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Contrary to average expectation, aureomycin given in adequate dosage is not very effective in permanently sterilizing the urine of patients with chronic prostatitis. Schatten and Persky studied the results of this therapy in twenty patients (who had only chronic prostatitis and no other genitourinary pathologic disorder) and found permanently sterile urine in only one case. They also found that bacteriologic findings were not influenced by the addition of prostatic massage. The explanation of the "failure" lies in the fact that the antibiotic is present in the prostatic tissue and secretions in insufficient concentration. (*Richard A. Leonardo, M.D.*)

Traumatic Cyst of the Spleen

A. David Schwartz, M.D., *Spring Valley, New York*

From the Surgical Service, United States Army Hospital, Fort Lee, Virginia.

Cyst of the spleen is not a common anomaly. It may easily be overlooked because of misleading symptoms and lack of definitive, localizing signs. The diagnosis may be difficult unless splenic palpation is carefully employed upon routine physical examination.

The purpose of this paper is to present the case report of a patient suffering from a traumatic cyst of the spleen in which medical aid was sought because of persistent back pain. This occurred four years after injury was sustained.

Unlike cysts of the liver, kidney, ovary or other abdominal viscera, cysts of the spleen are rarely encountered. The first splenic cyst recorded was discovered by Andral while performing an autopsy in 1829. The first successful removal of a splenic cyst was by Pean in 1867. In 800 splenectomies, only four cases were reported by Pemberton,[1] an incidence of 0.5 per cent. In the period from 1904 to 1940 only eight cases were found in the records of the Pathology Department at Bellevue Hospital by Sweet.[2] Between 1904 and 1934 the Mayo Clinic reported[3] 646 splenectomies of which only two were for hemorrhagic cysts.

Of the many classifications, Fowler's system of grouping splenic cysts[4] is perhaps the best: Primary or true cysts include congenital, traumatic, inflammatory, neoplastic (dermoid, epidermoid, lymphangioma and cavernous angioma) and parasitic (ecchinococcus) cysts; secondary or false cysts are those involving trauma, inflammation (tubercle bacillus, perisplenitis and the like) and degeneration (infarction secondary to thrombosis, embolus, endarteritis, ruptured aneurysm and the like).

Primary cysts have a secreting epithelial lining and secondary cysts do not; secondary cysts, approximately five times as common as primary cysts, are large, solitary and unilocular in 80 per cent of the cases. Echinococcus cysts occur about twice as frequently as all the non-parasitic cysts.

The etiologic picture of non-parasitic, splenic cysts is often unclear. Cysts have been found in young children, but are mostly prevalent between the ages of twenty and fifty. Men are less affected than women. McClure and Altemeier[5] believe that trauma plays the important role in the formation of large hemorrhagic or serous types, although they may occur spontaneously without a history of injury. These splenic tumors are described as encysted hematomas due to an injury which does not lacerate the surface of the spleen but which tears vessels in the interior of the organ, causing hemorrhage. With the passage of time, a cyst containing blood-stained fluid and fibrous walls is formed. If the blood is completely absorbed, the hemorrhagic cyst is converted to one of the serous type.

Cysts of the spleen, especially small ones, are often asymptomatic and may be found accidentally by the patient. If they cause no discomfort, the only subjective complaint may be a sense of heaviness in the left portion of the hypochondrium. Pressure on adjacent organs may cause symptoms not usually associated with splenic disturbances. Lang[6] reported a case in which the patient had pain in the chest, difficulty with bronchial breathing and a pleural effusion requiring thoracentesis. Dragging pain may be referred to the left shoulder, the left hypochondrium, the epigastrium, or the mid-back, as in the case presented herewith. Gastrointestinal disturbances, including constipation, flatulence, nausea and vomiting have also been described. If twisting of the splenic pedicle occurs, the patient almost immediately collapses. Palpation of the abdomen in all these cases will reveal a nontender tumor in the left upper quadrant, extending medially beneath the costal margin. Fluoroscopy reveals a high left leaf of the diaphragm with limitation of respiratory excursion. A flat plate of the abdomen reveals a large, left upper quadrant, soft tissue mass, pushing the transverse colon and stomach downward to the right. There may be bulging

on the left side of the lower intercostal interspaces and obliteration of the psoas shadow. The kidney is displaced downward on intravenous pyelography.

With no typical blood picture and such widely different signs and symptoms, a cyst of the spleen must be differentiated from cystic disease of the pancreas, omentum, mesentery, left lobe of the liver, ovary or kidney and other causes of splenomegaly. After arriving at a preoperative diagnosis of cyst of the spleen, the echinococcal type should be ruled out by means of the cutaneous allergic test of Casoni, the complement fixation test of Weinberg and Parvu, or the precipitin test of Welch and Chapman.

The treatment of choice is splenectomy, which has a mortality rate of 4 per cent.

CASE REPORT

An eighteen year old white woman was admitted to the United States Army Hospital, Fort Lee, Virginia, on October 29, 1953, complaining of a backache of four weeks' duration. The pain was localized in the thoracolumbar region and was non-radiating. Its onset had been gradual. The pain was dull and aching and was present when the patient sat or stood for any length of time. It was relieved by the patient resting in the supine position. This WAC private stated that four years previously she had sustained a mild cerebral concussion while riding in a bus. She had been kept in bed for a period of two weeks. She recalls no abdominal symptomatology at that time.

On physical examination of the abdomen, a smooth-surfaced, oval mass was revealed projecting below the left portion of the costal margin. It was firm, non-tender and ballottable.

Laboratory data indicated the following: urinalysis negative, white blood cells 6,000, Schilling differential within normal limits, hemoglobin 14.0 gm., sedimentation rate 5 mm. an hour, platelets 216,000, reticulocyte count 0.6 per cent, serology negative, bilirubin direct immediate 0.2 mg per cent, bilirubin total 0.7 mg per cent, urea nitrogen 7.0 mg per cent, prothrombin time 20 seconds.

A laparotomy performed on November 9, 1953, revealed a large, smooth-walled cyst, covered by a glistening capsule and attached to the inferior pole of the spleen. No accessory spleens were seen. Splenectomy and removal of the attached cyst intact were done through a high left, vertical, rectus-retracting incision. The spleen measured 10 by 5½ by 2 cm. in size. The fibrous-walled cyst, 16 by 12 by 10 cm., consisted of 960 cc. of yellowish fluid containing crystals of cholesterol. Sections of the spleen showed no gross pathologic change.

When the patient was examined six months after the operation, she had no symptoms. Her complete blood count, platelet count, prothrombin time and coagulation time were all within normal limits.

SUMMARY

A review of the history of cysts of the spleen, including an illustrative case report of a traumatic splenic cyst, is presented. The importance of palpation of the spleen upon routine physical examination is emphasized.

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A Complication Following the Use of a Ureteral Stone Extractor

FRANCIS A. BENEVENTI, M.D., *New York, New York,* AND FRANCIS S. CREIGHTON, M.D., *Elmira, New York*

From the Joseph McCarthy Clinic of the New York Polyclinic Medical School and Hospital, New York, New York.

THE purpose of this paper is to report the interesting events which took place when we attempted to remove a small stone from the ureter with a stone extractor (basket).

FIG. 1. Excretory urogram showing the obstructing stone in the right lower ureter with urinary back pressure on the right upper tract. Note the dilated calyces and pelvis. The left side is normal.

CASE REPORT

About four days before we first saw this short, obese, sixty-five year old woman, she complained of radiating right flank pain. There was some nausea and vomiting, and definite flank tenderness but no elevation of temperature. Numerous red blood cells were found in the urine. All other findings were irrelevant. A plain x-ray film of the abdomen showed an opaque shadow measuring 0.5 by 1.0 cm. in the region of the right upper ureter at the level of L4.

Three days later another plain x-ray film of the abdomen plus an excretory urogram showed that the previously reported stone had descended to the lower ureter and was located 5 cm. proximal to the bladder juncture. There was definite evidence of urinary back pressure as manifested by the dilation of the calyces and pelvis of the right kidney. (Fig. 1.) When the patient's temperature rose to 105°F., she was referred to us. We decided to relieve the obstruction in the lower ureter by cystoscopic means. Under light pentothal® anesthesia a No. 5 F. catheter could be advanced only 5 cm. up the right ureter when it met an impassable obstruction. The catheter was then withdrawn and a stone extractor with its usual filiform tip was introduced into the right ureter; to our amazement it by-passed the stone. By opening the basket we were able to engage the calculus. (Fig. 2.)

Thus far all was well, but we soon discovered that we could not withdraw the instrument by gentle traction. After several attempts at trying to remove basket and stone, the patient was returned to her room where continuous 1 pound pulley traction on the basket was maintained over the foot of the bed. Her temperature dropped to normal within hours and she was perfectly free of pain and symptoms except for the inconvenience of having to remain flat in bed. After seventy-two hours of futile continuous traction it was decided to perform pelvic ureterotomy to remove the incarcerated basket and stone. X-ray showed that the tip of the instrument had become unscrewed and was lying free in the lower ureter and that the stone had dropped out of the basket and was lying between the loose filiform tip and the wire basket. (Fig. 3.)

Through a right rectus incision the lower ureter was opened, and the loose unscrewed

2

3

FIG. 2. Basket in place in the right lower ureter.

FIG. 3. The tip of the basket has become unscrewed and is lying free in the ureter.

catheter tip and the stone were removed quickly and easily. The treaded tip of the basket was grasped with artery forceps but it could not be drawn up into the ureter where we had planned to remove it by using a wire cutter. The bladder was next opened, and through this cystotomy opening a generous superior meatotomy was made in the right ureteral orifice. However, even with this additional exposure and loosening, the basket could not be mobilized. We then divided the posterior wall of the bladder down to the ureter and found the basket hopelessly enmeshed in an edematous, strictured lower ureter. The basket was lifted out with some difficulty, divided with wire cutters and removed. Now, however, we were left with a hopelessly strictured lower ureter which could only produce further obstruction if allowed to remain. This, of course, was of no possible use to the patient.

In freeing the right ureter we found that we could gain an extra length of ureter. Fortunately this made it possible to sacrifice the distal 2.5 inches and still have enough ureter left over to reimplant into the bladder without tension. This was done, and a soft rubber splinting catheter was brought out through the cystotomy opening and allowed to remain in place for eighteen days. The patient made an uneventful recovery.

SUMMARY

A case is reported in which stone extraction by cystoscopic means was attempted in a patient with an obstructing stone in the lower ureter, producing fever and back pressure. The extractor became incarcerated in the lower ureter and its filiform tip became unscrewed. On pelvic ureterotomy the instrument tip and stone were easily removed but not the basket. Cystotomy and superior meatotomy were of little help. It was finally necessary to divide the posterior wall of the bladder down to the trapped instrument to remove it. The distal 2.5 inches of ureter were excised and the ureter was successfully reimplanted into the bladder.

BOOK REVIEWS

A Manual on Cardiac Resuscitation. By Robert M. Hosler, M.D., F.A.C.S. Foreword by James H. Rand, III, D.SC. (Hon.). Introduction by Claude S. Beck, M.D. Cloth binding, 183 pages, illustrated. Springfield, Illinois, 1954. Charles C Thomas, Publisher. *Price* $4.00.

This small book should be in every hospital library or in the hands of the surgeon or surgeons who spend their working hours in the operating room. One has only suddenly to be faced with a case of cardiac arrest in a patient being subjected to surgery in order to comprehend the frightful mental shock the operator, the anesthetist and the surgical assistants experience. When this calamity occurs no time can be lost. Someone, within a matter of seconds, must know how to meet this emergency and have the necessary tools and drugs at hand if a life is to be saved. Unless the stage is always set and the operator knows his "stuff" in detail, death will be the end result. Dr. Beck writes, "It [this book] presents information that should be known before and not after the crisis occurs. The time should be past when we surgeons turn these emergencies over to someone else to take care of. We should take care of them ourselves."

This short monograph should be thoroughly studied by everyone—surgeon, assistants, anesthetist and nurses—dealing with surgery.

Illustrated Review of Fracture Treatment. By Frederick Lee Liebolt, B.A., M.D., D.SC., LL.D. First edition, 229 pages, 605 illustrations. Los Altos, California, 1954. Lange Medical Publications. *Price* $4.00.

A compact, instructive work, sans padding, on the treatment of fractures. Although the text covers, with a minimum of detail, what most textbooks cover in endless pages, it is the illustrations (the roentgenographs in halftone and the others in line cuts) that are the outstanding and valuable features. They clearly show what they were drawn or photographed to show. Every known fracture is covered. Young surgeons should become familiar with such basic knowledge as the clinical examination of fractures and principles of treatment of fractures. The author informs the reader that "this review is intended to illustrate and to discuss, briefly and systematically, the principal features concerning the diagnosis and treatment of fractures. It has been written for the medical student, the house officer, and the general practitioner. . . . It is to serve as a supplement to the standard texts in the field."

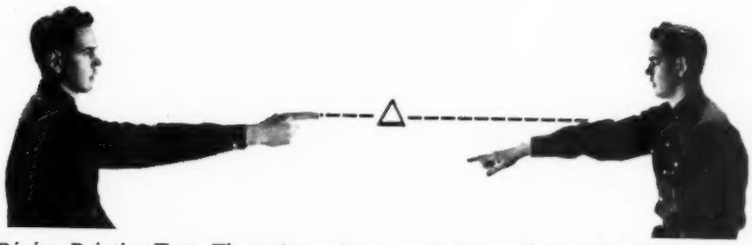
Anatomy of the Bronchovascular System. Its Application to Surgery. By George L. Birnbaum, M.D. Foreword by Evarts A. Graham, M.D., 300 pages, 99 figures. Chicago, 1954. Year Book Publishers, Inc. *Price* $15.00.

Dr. Graham in his Foreword tells us the thorax is the last region of the body to be opened to deliberate surgical attack. Also, although occasional successful operations were performed in the field long ago, it was the first World War which furnished the incentive for the development of planned intrathoracic procedures. Between the two wars amazing advances have been made. Dr. Birnbaum and his colleague, Dr. Pol Coryllos, played important roles in many of the fundamental advances. We learn that postpneumonic empyema has become almost obsolete, pulmonary abscess is becoming less frequent, and bronchiectasis is gradually being reduced to the vanishing point.

The author states that originally it was his purpose to prepare a modest line-drawing atlas depicting most of the usual and some of the unusual patterns of the pulmonary arteries and veins. Imperceptibly this led to other fields; and before he was finished, this work was born.

Chapter 1 covers the Embryology of the Bronochovascular System; Chapter 2, Anatomy of the Bronchovascular System; Chapter 3, Anomalies of the Pulmonary Vessels; Chapter 4, Surgery of the Pulmonary Vessels; Chapter 5, Tracheobronchial Surgery; Chapter 6, Excision of Pulmonary Tissue.

The illustrations are excellent. There are selected drawings from an original atlas of pulmonary arteries and veins. This is a well planned and superbly written book—an important book on this branch of surgery—and should enjoy a wide distribution.



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2. The Caloric (Bárány) Test. *The patient sits with his eyes fixed on a stationary object and the external ear canal is irrigated with hot (110 to 120 F.) or cold (68 F.) water. If the vestibular nerve or labyrinth is destroyed, nystagmus is not produced on testing the diseased side.*

3. The Rotation (swivel chair) Test. *The patient sits in a swivel chair with his eyes closed and his head on a level plane. The chair is turned through ten complete revolutions in twenty seconds. Stimulation of a normal labyrinth will cause nystagmus, past pointing of the arms and subjective vertigo.*

Notes on the Diagnosis and Management of "Dizziness"

I. Vertigo

The term "dizziness" (vertigo) should be restricted to the sensation of whirling or a sense of motion.[1] This sensation is usually of organic origin and is the tangible symptom of a specific pathology.

Moderate vertigo, with a sense of motion and a whirling sensation, may be produced by infection, trauma or allergy of the external or middle ear. Examination of the ear will usually disclose the abnormality.

Severe vertigo, which will not permit the patient to stand and causes nausea and vomiting, indicates an irritation or destruction of the labyrinth. The specific condition may be labyrinthine hydrops, an acute toxic infection, hemorrhage or venospasm of the labyrinth or a fracture of the labyrinth. Multiple sclerosis and pathology of the brain stem should be considered also.

It is important to learn if the patient's sensation is continuous or paroxysmal.[2] Paroxysmal vertigo suggests specific conditions: Ménière's syndrome, cardiac disease and epilepsy. Continuous vertigo without a pattern may be due to severe anemia, posterior fossa tumor or eye muscle imbalance.

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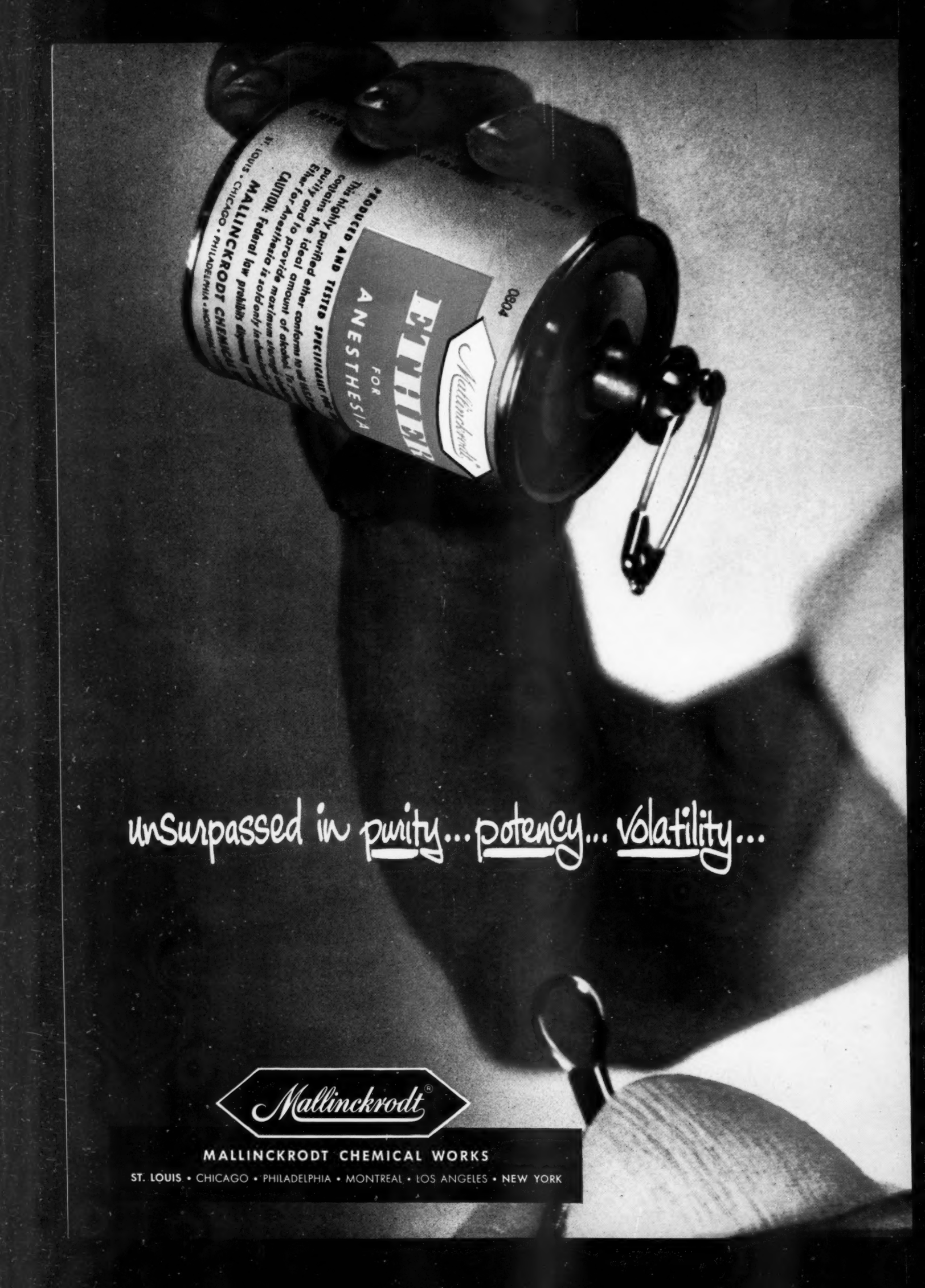
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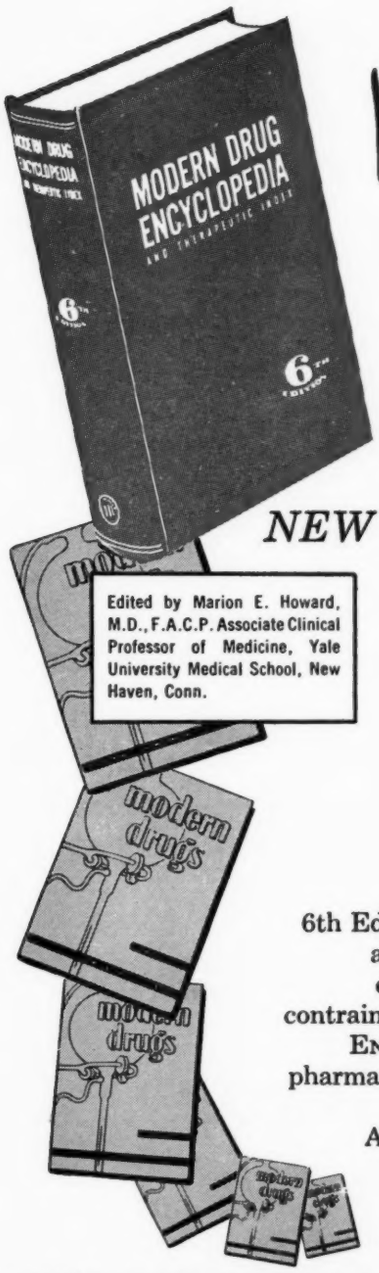

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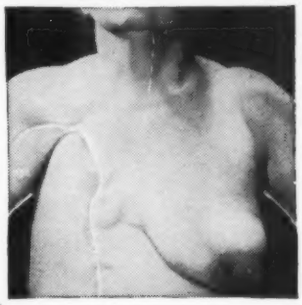

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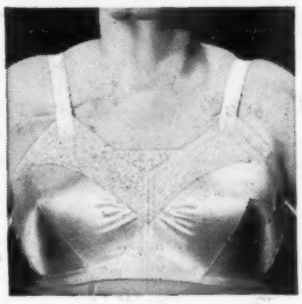

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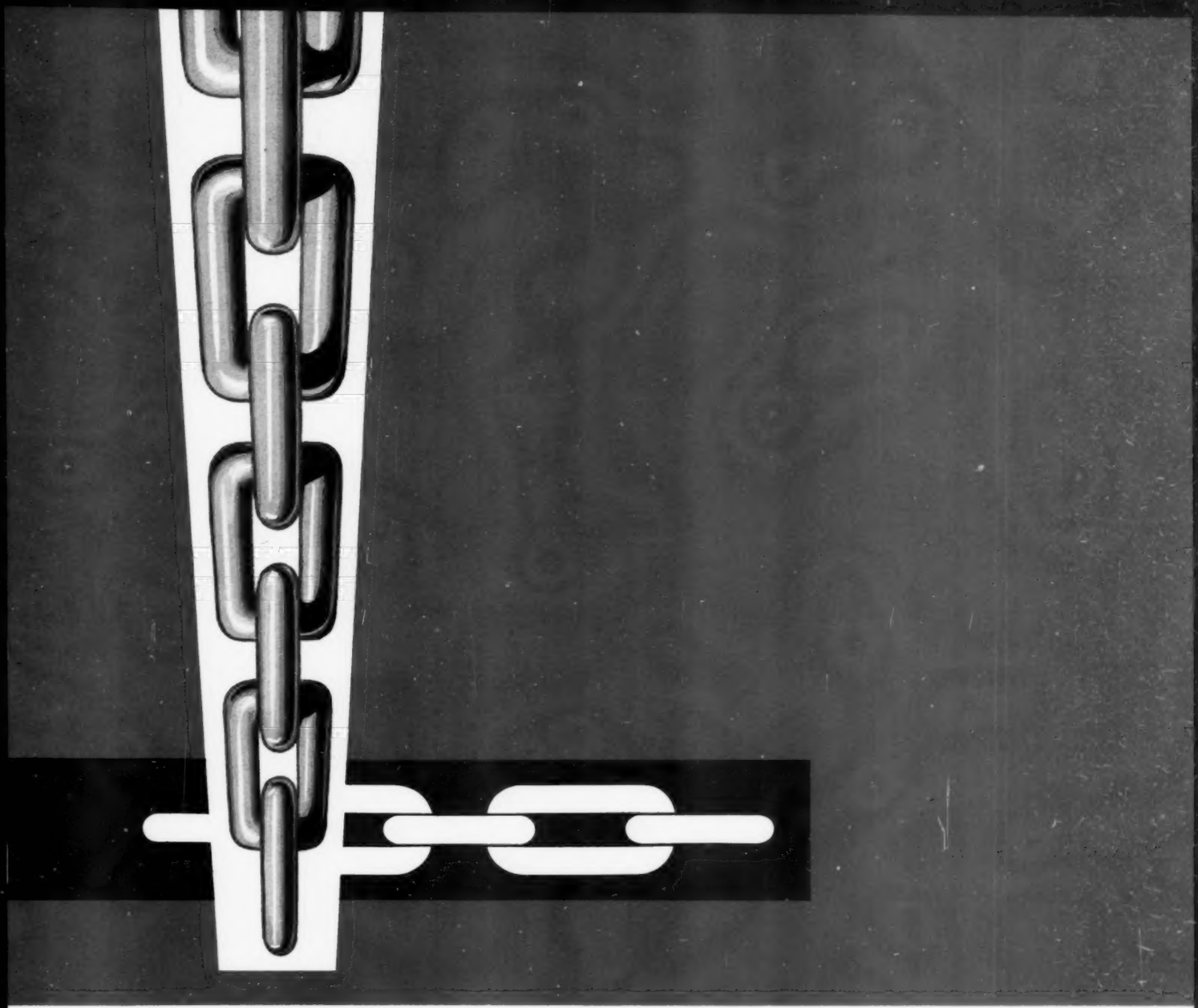

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POSTOPERATIVELY • to prevent seepage and hemorrhage

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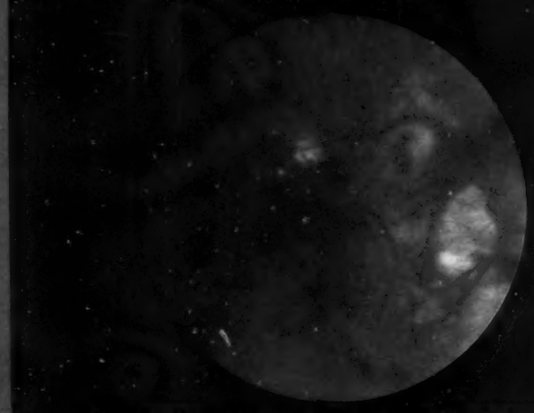
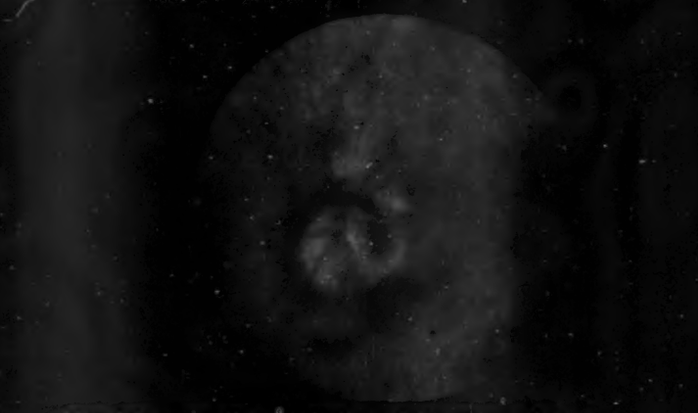
Welling up of blood upon incision of lid and conjunctiva, common in ophthalmic procedures, hampers surgery.

Minimal bleeding after Adrenosem therapy facilitates surgery and promotes healing.

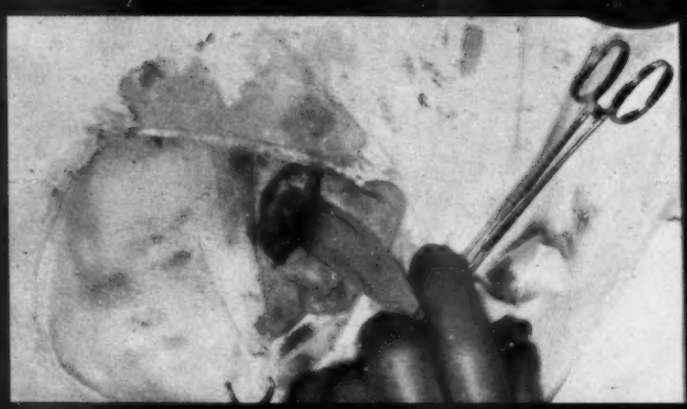


Anticipation of recurrent hemorrhage and visual defect is high in hypertensive patients.



After Adrenosem therapy. Note exudative areas. Maintenance therapy checks further hemorrhage.

Mastoidectomy with a bloody field obscures view, requires frequent suctioning.



After Adrenosem therapy. Note clearer operative field with minimized bleeding.

Multiple extraction without Adrenosem. Note seepage. Frequent suctioning is required.

After Adrenosem therapy. Note clearer field. Minimal bleeding facilitates surgery and promotes healing.

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Adrenosem is specific for conditions characterized by increased capillary permeability allowing oozing and seepage bleeding. To prevent seepage bleeding due to capillary permeability it is necessary to maintain tone of the capillary wall. This is supported by Pappenheimer's[1] work on capillaries. In cases of capillary permeability it is postulated that the connective material between the cells, which form the capillary wall, has lost its tone and allows the erythrocytes to pass through.

To confirm the action of Adrenosem in establishing capillary integrity the cheek pouch of a hamster was irritated with snake venom, which has the property of making the capillaries more permeable.[2] A great many petechiae appeared on the surface and a 500 X magnification showed the capillaries with extravasation of erythrocytes. Another animal was treated with Adrenosem, and 30 minutes later, the same amount of snake venom was used to irritate the cheek pouch. Microphotographs of the same 500 X magnification show the intact capillary walls and the actual blood cells coursing through the capillaries, without signs of escaping erythrocytes.

Fig. 1—*Trauma from snake venom produces hemoconcentration and stasis, indicative of altered vascular permeability.*

Fig. 2—*Trauma does not produce petechiae following Adrenosem injection.*

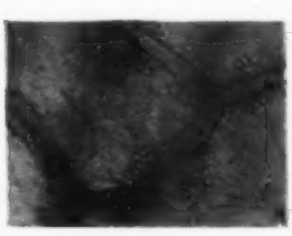

Fig. 1

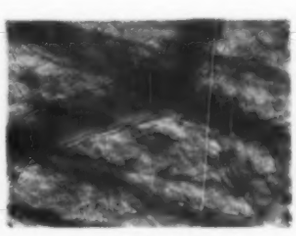

Fig. 2

Action

A systemic hemostat, Adrenosem restores capillary integrity by maintaining tone of intercellular tissue in the capillary wall. Adrenosem is unlike any hemostat available to medicine.[3,4] It does not interfere in any way with the blood clotting mechanism or its component parts. There is no sympathomimetic action and the blood pressure, cardiac rate and volume are not affected. Adrenosem may be administered simultaneously with any type of anesthetic, anticoagulant or vitamin K and heparin. Adrenosem will not interfere with the action of these agents.

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While the hemostasic action of epinephrine and related compounds has been recognized for a long time, certain characteristics limited their usefulness. A product of the metabolism of epinephrine seemed to offer the most promise. Given the name, Adrenochrome, this compound was studied extensively in both European and American centers. The handicap to wide use of Adrenochrome was its instability, so efforts were directed to find a stable modification that would retain the physiological activity. The answer to this proved to be the monosemicarbazone of Adrenochrome. Further research demonstrated that this compound could be made soluble enough to be practicable as an injectable solution, by transforming it to a sodium salicylate complex. This process has made possible Adrenosem Salicylate, a potent therapeutic agent which fills an important need in medicine.

1 *Pappenheimer, John R.: Passage of molecules through capillary walls, Physiol. Rev. 33:387-423, 1953.*
2 *Fulton, M. D., Department of Biology, Boston University: Personal communication, 1954.*
3 *Sherber, Daniel A.: Adrenochrome complex in the control of bleeding, Am. J. Surg. 86:331-335, 1953.*
4 *Roskam, J.: Arrest of Bleeding, Chas. C. Thomas, Publisher, Springfield, Ill., 1954.*
5 *Peele, J. G.: Adrenosem in the control of hemorrhage from the nose and throat (in press).*
6 *Bourgoyne, J. R. and Riddle, A. C., School of Dentistry, University of Tennessee: Personal communication, 1954.*

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- preoperatively to control oozing during surgery and provide clearer operative field: one to two ampuls (5 to 10 mg.) every two hours prior to surgery for two doses.
- postoperatively to prevent hemorrhage and check oozing: one to two ampuls (5 to 10 mg.) every two hours until there is no indication of undue bleeding. To maintain control, one ampul may be administered every three hours or 1 to 5 mg. orally t.i.d.
- to control active bleeding: one ampul (5 mg.) every two hours until bleeding is controlled; frequency of dose may then be diminished.
- to control mild, low-grade bleeding: one ampul (5 mg.) every three or four hours until bleeding is controlled; then 1 to 5 mg. orally four to five times daily until bleeding ceases.
- maintenance dosage to prevent bleeding in conditions where small vessel integrity may be impaired: 1 to 5 mg. orally t.i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally every three or four hours; if bleeding persists, oral dosage should be supplemented with one ampul (5 mg.) daily.
- pediatric dosage: up to four years of age—1 mg. intramuscularly or orally with same frequency as for adults, until bleeding is controlled. From four to twelve years of age—one-half the adult dosage as indicated.

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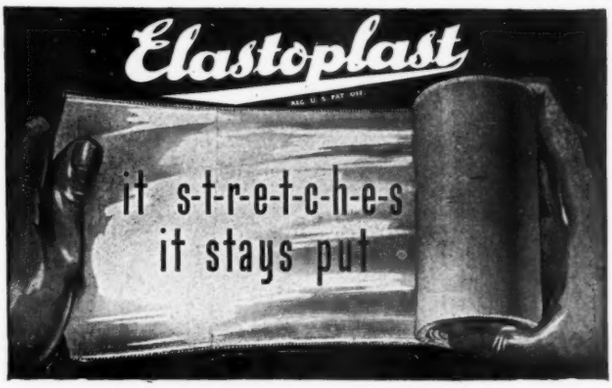

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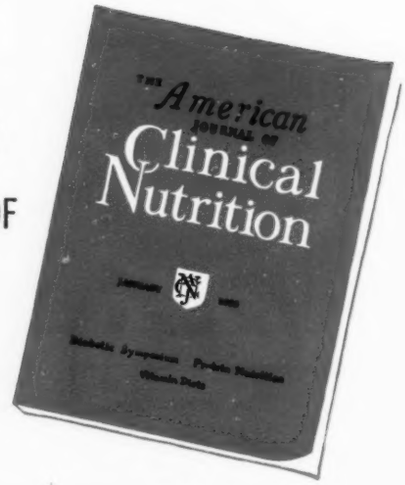

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NOTES AND QUOTES

According to popular belief, the only student acceptable to the medical schools of the United States is one who has taken practically nothing but science courses, made straight A's, captained the football team, led the student council and made a 99 per cent score on the Medical College Admission Test. In the February issue of The Journal of Medical Education, *Dr. Daniel H. Funkenstein, clinical associate in psychiatry at Harvard Medical School, described these "qualifications" as misconceptions.*

Dr. Funkenstein described how students who plan their college career on the basis of these erroneous impressions often miss the intangibles which contribute so much to personality growth and maturity. They become tense and overly competitive, losing out on the feelings of cooperation and comradeship which can aid the learning process and make college a more rewarding experience.

The article listed statistics which show that, at Harvard, the admission procedure runs counter to these standard myths about medical schools. Students with a B average or less comprise 20 per cent of the admissions, and the average score on the Medical College Admission Test for Harvard students was at the 84th percentile. The article also stated that 17 per cent of the entering class received a recommendation from their colleges that described them as less than excellent, and 29 per cent were placed by the Harvard interviewers in less than the top category.

Furthermore, although a majority of the students accepted are science majors, Dr. Funkenstein points out that many non-science majors are also enrolled, and that the admissions committee often looks askance at a man who has piled "science course on science course" at the expense of a broad education.

Actually, what Harvard is looking for in a prospective medical student is a mature man with a broad education. As medicine shifts from the "patient as a disease" concept to the "patient as a human being" concept, the outstanding doctor of the future will be the one with vision, creative imagination, insight and emotional maturity.

Dr. Funkenstein feels that admission committees should consider these qualities as of primary importance, rather than arbitrary standards of grades, extracurricular activities or the number of science courses taken.

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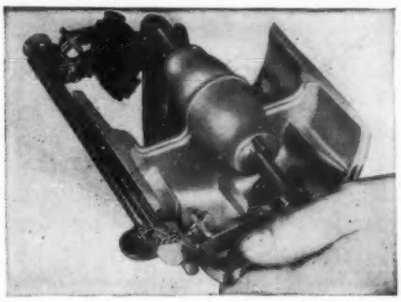

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The service is primarily designed for members of the Association but the Association will permit non-members (especially those who have never written for publication before) to submit two manuscripts for editing at 50 per cent increase in the above rates. About ten days' time will be required to edit a manuscript.

All manuscripts should be sent to: American Medical Writers' Association, Harold Swanberg, B.S., M.D., F.A.C.P., Secretary, 209–224 W.C.U. Bldg., Quincy, Ill.

A leaflet giving further details of the service will be gladly sent on request.

Baylor University Hospital in Dallas, Texas, was the recipient of a gift of $75,000 for the purchase of a "Theratron" Cobalt 60 Beam Cancer Therapy unit. The gift was made in January, 1955, by the Reserve Life Insurance Company of Dallas, Texas in honor of the company's President, C. A. Sammons. The installation of this advance machine will place Baylor among the leading centers for treatment of cancer in the United States.

The unit, which weighs 8 tons, will require a special building with heavy lead and concrete insulation on ceiling, walls and floor to keep radioactivity under positive control.

The new cancer therapy unit and the first supply of atomic radiation is scheduled for delivery to Baylor in June, 1955. The building and facilities, which will be valued at $150,000.00, will be known as "The Charles A. Sammons Irradiation Therapy Center."

THEODORE G. KLUMPP, M.D., President of Winthrop-Stearns Inc., has been appointed a member of the National Advisory Council on

CONTINUED ON PAGE 50

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NOTES AND QUOTES

Vocational Rehabilitation, a new Federal agency organized to restore the nation's handicapped to useful lives.

The appointment was made for a four-year term by Nelson A. Rockefeller, Acting Secretary of the Department of Health, Education and Welfare, under terms of the Vocational Rehabilitation Act of 1954. Enacted unanimously by both Houses of Congress, the Act is the second to be passed in President Eisenhower's four-point national health program.

At Morris Plains, N. J., ground was broken recently for a new building to expand the research facilities of Warner-Chilcott Laboratories, the pharmaceutical division of Warner-Hudnut, Inc. According to an announcement by E. Roth Janes, Vice-President, and Dr. George H. Mangun, Director of Research of Warner-Chilcott, the building addition will quadruple the space now available for the division's various research projects. Dr. Mangun stated that a number of current research programs would be aided by the expanded facilities. Among these he listed the study of hormones and enzymes, both of importance in geriatrics as well as other areas of medicine; and research in pharmacology, physiology and virology.

THE CHALLENGE TO MEDICAL EDUCATION, a study of medical education in the United States, has been prepared by Public Affairs Pamphlets, Inc. A number of copies have been made available by The National Fund for Medical Education which is doing so much to help solve the serious financial problem confronting our medical schools. The pamphlet traces briefly the historical development of medical education in America and makes clear how important it is that we maintain the highest possible medical teaching standards. Only thus can we obtain better health services for all Americans.

The Atomic Energy Commission stated recently that medical and biologic information developed by its scientists on the effects of nuclear radiation, blast, flash burns and fallout is made public as rapidly as it can be properly evaluated and correlated. The Commission denied that the medical profession is refused access to the results of its studies of the effects of nuclear explosions on human beings and animals and methods for treating casualties. It is the Commission's long-standing policy that the American people and medical science should be informed, as rapidly as possible, of the biomedical aspects of atomic energy. A large amount of information of this nature has been made public and the Commission's report of February 15th on the effects of high-yield thermonuclear explosions declassified much additional information.

THE tenth annual Schering Award competition for medical students opened recently, as announced by Dr. Robert W. Burlew, Chairman of the award committee. Its aim is to encourage medical writing and exploration of current research literature.

Titles of three subjects on which American and Canadian students are invited to submit papers this year are: Current Concepts in the Management of Osteoporosis; Prevention and Treatment of Blood Transfusion Reactions; and Recent Trends in the Clinical Use of Adrenocortical Steroids.

Both a $500 first prize and a second one of $250 will be awarded for each of the three subjects. Outstanding authorities in each field will select the winners. Special recognition in the form of a professional gift will be given each student submitting a meritorious paper.

Deadline for entry forms specifying the choice of title is July 1st. Manuscripts should be mailed not later than October 1st. Students may compete individually or cooperatively in teams. Information and instructions for the award competition are available from the Schering Award Committee, 60 Orange Street, Bloomfield, N. J.

The American Chemical Society's Garvan Medal, honoring women in chemistry, was presented at Cincinnati, Ohio, recently to Dr. Grace Medes, senior member of the Institute for Cancer Research and the Lankenau Hospital Research Institute, Philadelphia. Dr. Medes, who is internationally known as a leader in research on the chemistry of life processes, received the gold medal at a general assembly of the Society in Cincinnati's Netherland Plaza Hotel during the Society's 127th national meeting.

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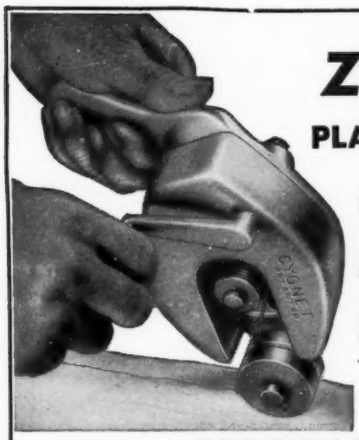

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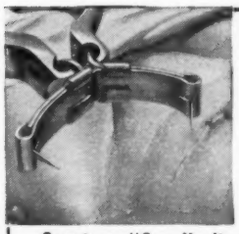

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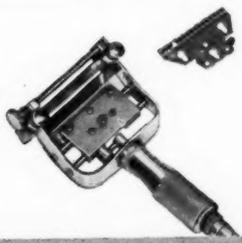

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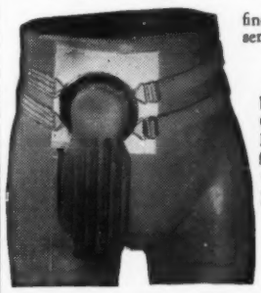

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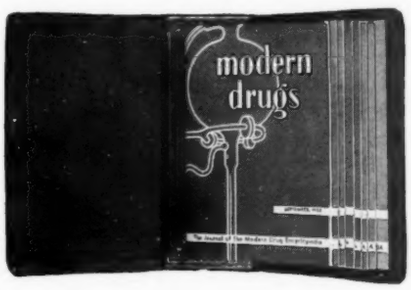

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NOTES AND QUOTES

THE next scheduled oral and clinical examinations (Part II) for all candidates for the American Board of Obstetrics and Gynecology will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from May 11 through May 20, 1955. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

Candidates who participated in the Part I examinations will be notified of their eligibility for Part II as soon as possible. For further information contact: Dr. Robert L. Faulkner, Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

Sulfadiazine is the best prophylactic against the threat of urinary tract infection following abdominal operations when antibiotics are not required for a specific reason, Dr. J. Englebert Dunphy, Clinical Professor of Surgery at Harvard Medical School reported recently in The Practitioner.

"*Certain operative procedures in the abdomen predispose to urinary-tract infection,*" *Dr. Dunphy warned in discussing prophylactic uses of antimicrobial agents in abdominal surgery.* "*Initially, if a combination of streptomycin and penicillin is used,*" (*to provide intestinal antisepsis*), "*this will suffice to protect the urinary tract. Later, when these antibiotics may not be necessary for other reasons, one of the sulfonamides may be continued in small doses orally. This will produce a definite bacteriostatic effect. Sulfadiazine remains the most effective agent for this purpose.*"

THE American people spend five times more for liquor than for medicines and other drug store items, according to Dr. Theodore G. Klumpp, President of Winthrop-Stearns Inc., pharmaceutical manufacturer. Dr. Klumpp is Chairman of the Hoover Commission Task Force on Medical Services, which just submitted a report of its study to Congress. The annual outlay for medicines per person is only $10.00, or "the same amount that goes for spectator amusements," he told the Michigan Clinical Institute. Actually, the cost of health products today is small "in terms of lives saved, diseases conquered or controlled, shortened duration of illness and over-all expense to the patient." Achieving these goals has largely been due to the remarkable advances of medicine, pharmacy and the drug manufacturing industry, according to Dr. Klumpp.

Referring to health advances, he said more progress has been made in the twentieth century in the conquest of disease and prolongation of life than in "the entire history of man's previous existence on earth." Life expectancy has increased from forty-nine years in 1900 to almost sixty-eight in 1953 and the infant mortality rate has dropped from 162 per 1,000 births to 30 per 1,000. Many killer diseases have been almost completely eliminated and "others have lost their sting."

The Jesse H. Jones Fellowship in Cancer Education honoring Dr. E. W. Bertner was awarded recently to Dr. Melvin L. Samuels at the banquet of the 9th Annual Symposium on Fundamental Cancer Research, sponsored by The University of Texas M. D. Anderson Hospital and Tumor Institute. Dr. Samuels, a graduate of Villanova University and Jefferson Medical College in Philadelphia, was resident in pathology at Baylor Hospital in Dallas. He will be in the Department of Medicine at Anderson Hospital.

The Cancer Education fellowship honoring Dr. Bertner, the first acting Director of Anderson Hospital and the first President of the Texas Medical Center, was established in 1950 by Houston Endowment, Inc., a philanthropic organization established in 1937 by Mr. and Mrs. Jesse H. Jones for the support of any charitable, educational or religious undertaking.

THE 28th Annual Scientific Session of the American Heart Association will be held on October 22, 23 and 24, 1955, at the Jung Hotel, New Orleans, Louisiana. Meetings sponsored by the Council on Community Service and Education and the Council on Rheumatic Fever and Congenital Heart Disease will be held on October 23rd and 24th. The Assembly Panels will follow the Scientific Sessions on October 25th and the General Assembly will be held on the morning of October 26th.

Other important events during the meeting include the Annual Dinner of the American Heart Association on the evening of October 24th and the Assembly Luncheon on the 25th.

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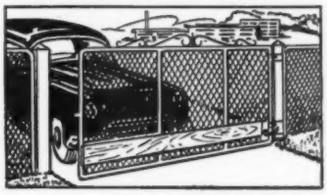

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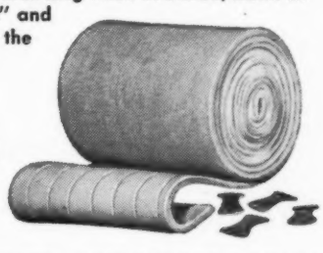

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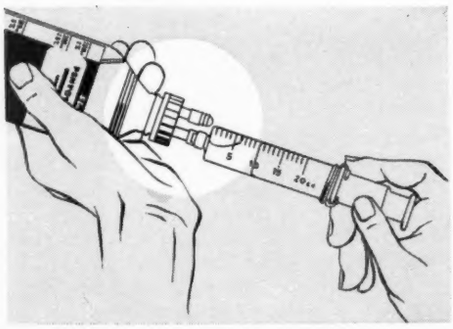

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